The Royal Society
of Canada

LEAD IN THE CANADIAN ENVIRONMENT: SCIENCE AND REGULATION

FINAL REPORT

THE COMMISSION ON LEAD IN THE ENVIRONMENT

SEPTEMBER 1986

OFFICE OF THE PRESIDENT

CABINET DU PRÉSIDENT

THE ROYAL SOCIETY OF CANADA
LA SOCIÉTÉ ROYALE DU CANADA

September 15, 1986

The Hon. Thomas McMillan, P.C., M.P.
Minister of the Environment
Environment Canada
Terrasses de la Chaudiere
Ottawa, Ontario
K1A OH3

Dear Mr. McMillan:

I have the honour to present to you, in accordance with the terms of our contract with your Ministry, the final report of the Royal Society of Canada Commission on Lead in the Environment.

The main recommendations and conclusions are summarized in the Chairman's Appraisal on pp. xix-xlvi. You will see at once that the Commission's recommendations, if found useful, will require action by several other Ministers, and also by your colleagues in the Provincial governments. We have not, however, presumed to direct any recommendation to anyone but yourself.

The terms of our contract call for the Commission to remain in being until September 1st, 1987, so as to respond to comments or criticisms. We intend to honour this request, but propose to close the offices of the Commission, and to release its excellent staff, by December 31st, 1986. Any further business after that date will be dealt with by the Chairman and Manager, who will remain at your disposal.

With some pleasure I confirm that this work has been carried out within the span of time foreseen, and within the original budget.

Yours sincerely,

Alexander G. McKay.

Alexander G. McKay, F.R.S.C.
President

344 Wellington Street
Ottawa, Ontario
K1A ON4

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COMMISSION ON LEAD IN THE ENVIRONMENT
COMMISSION D'ÉTUDE DU PLOMB DANS L'ENVIRONNEMENT
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September 10, 1986

Dr. Alexander G. McKay, F.R.S.C.
President
The Royal Society of Canada
344 Wellington Street
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Dear Mr. President:

I have the honour to present to you the final report of the Commission on Lead in the Environment. It has been reviewed in accordance with the Society's policies.

Several other reports will be completed before December 31st, 1986, the date on which I propose to close the Commission's offices, and to release the staff. These will include two major scientific overviews, entitled "Health Effects of Lead" and "Pathways, Cycling and Transformation of Lead in the Environment". I hope that the Society will consider publishing these large works as Memoirs.

We shall also present a bibliographic summary, and a further volume containing the major studies by outside agencies financed as requested by the Commission. You will recall that we have already published an interim report, "Lead in Gasoline: a Review of the Canadian Policy Issue," and a supplementary report, "Alternatives to Lead in Gasoline".

All the Commissioners join with me in assuring you that their work would never have been completed--in fact would barely even have been started--without the superb work of its tiny staff. The Society owes them a deep debt of gratitude for a job done far beyond any call of duty.

Yours sincerely,

F. Kenneth Hare, F.R.S.C.
Chairman

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Acknowledgements

A broad-ranging inquiry such as that conducted by the Royal Society of Canada Commission on Lead in the Environment needs the help of many individuals and organizations. We risk being invidious by attempting to single out any for special recognition. The following, none the less, must be mentioned:

- the individuals, government departments and agencies, interest and neighbourhood groups who took the trouble to prepare and submit briefs
- the individuals and organizations who generously provided reference material
- the industries that arranged site visits, namely, Esso Petroleum, Cominco, Noranda, Brunswick Mining and Smelting, Canada Metal Company and Toronto Refiners and Smelters
- those who organized or conducted workshops on behalf of the Commission, namely, PACE, ILZRO, DuPont Canada Inc., Ethyl Canada Inc. and the Canada Metal Company Ltd. (with the co-operation of various representatives of the metal industry)
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- the Director-General and research scientists of the Institute for Ocean Sciences, Patricia Bay, BC
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- Dr. Arthur Chamberlain, formerly of the Environmental and Medical Sciences Division of the Atomic Energy Research Establishment, Harwell, UK
- the staff of the Environmental Protection Service (Administration and Finance Section of Environment Canada) who responded so effectively to the special needs of the Commission
- the army of reviewers and other individuals who read the text of the Final Report, and made helpful suggestions

Terms of Reference

Purpose

To provide the Minister of the Environment with independent advice on the present and future risks and areas of concern resulting from the presence of lead in the Canadian environment, and

To consider and propose, as may be required, corrective measures to reduce the environmental and health risks associated with lead which may be adopted either independently by the Minister of the Environment or in co-operation with others. The effects of lead in the workplace are specifically excluded from these terms of reference.

Considerations

To fulfil these objectives the Commission is to report on the following:

1. (a) the sources of lead releases in Canada and their relative contributions;

   (b) the pathways by which lead enters the Canadian environment and the means and media by which lead is transported within the environment and to humans;

   (c) the toxicity of lead;

   (d) the potential or actual exposure of, and risks to, human and environmental targets in Canada;

2. (a) practical corrective measures, as may be required, to reduce the risks associated with lead based on the Commission's review and assessment of the impact and implications of lead for the environment and for humans;

   (b) the economic, technical, social (where appropriate) and labour implications of reductions in lead releases and exposure from all sources, including the implications of eliminating lead in gasoline.

Duration and Reporting

The Commission will sit for three years and provide a progress report by September 1, 1985, on the direction and findings of its review, and a final report including its recommendations by September 1, 1986. The Commission shall continue to sit for a third year to receive and review comments on its reports and to make recommendations on those comments.

The Commission's progress and final reports will be made available to the public.

All written submissions made to the Commission except those deemed by the submittor to contain confidential business information will be made available to the public.

Commissioners, Consultants and Staff

Commissioners

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Friends of the Earth
Niagara Neighbourhood Association
South Riverdale Community Health Centre

The Commission thanks its consultants for their help. It has been influenced by their advice, but has not always taken it.

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WILLIAM H. GAUVIN is President of William H. Gauvin Technologies Inc., and Auxiliary Professor of Chemical Engineering, McGill University. He was Director of Research and Development, Noranda Mines, Ltd., 1970-1982. He spent two terms on the Science Council, served on the Board of McGill University (1972-82) and is a former President of the Chemical Institute of Canada. He is a Companion of the Order of Canada, and a Fellow of the Royal Society of Canada.

F. KENNETH HARE is former Provost of Trinity College in the University of Toronto, where he is University Professor Emeritus in Geography. In 1972-1973 he was Director-General of Research Coordination in Environment Canada. At various times he has chaired Canadian enquiries into nuclear waste disposal, acid deposition and nuclear winter (chiefly for the Royal Society of Canada, of which he is a Fellow). He is an Officer of the Order of Canada, and President of Sigma Xi.

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LUCIANO C. NERI is Professor of Epidemiology and Community Medicine, University of Ottawa, and a member of the NRS Sub-committee on Heavy Metals. He led a survey of lead levels in Trail, BC, in 1975, and has also worked in public health in Italy and Venezuela. He is a Fellow of the Royal College of Physicians and Surgeons of Canada, and a charter member of the Society for Environmental Geochemistry and Health.

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Staff

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Consultants

ARTHUR C. CHAMBERLAIN recently retired as a senior research scientist at the Atomic Energy Research Establishment, Harwell, UK. His work has included the use of radioisotopes as tracers in a number of fields, but has also been much broader in scope. Early studies included work on the dispersion of materials in atmospheric turbulence near the ground. He has specialized in recent years on the effects of toxic metals on human metabolism.

SERGIO FACCHETTI is chief of the Radiochemistry and Nuclear Chemistry Division at the Commission of the European Communities Joint Research Centre at Ispra, Italy, where he is Principal Investigator of the Isotopic Lead Experiment. He is Professor at the University of Pavia and is a member of the US EPA Committee on Air Quality Criteria for Lead. He holds the Gold Medal of the Italian Chemical Society, and has been awarded the United Nations Industrial Development Organization's Certificate of Merit.

DAVID HEWITT is Professor in the Department of Preventive Medicine and Biostatistics at the University of Toronto, and since 1975 has served as Coordinator of Graduate Studies in Community Health. His research has included studies of health hazards related to air pollution, obstetric radiography, uranium mining and lipid constitution. He is a founding Fellow of the American College of Epidemiology.

PETER L. ROSENBAUM is Professor in the Faculty of Health Sciences at McMaster University. His clinical, research and teaching interests are in developmental pediatrics, particularly with children with neuromotor disabilities and their families. Before his appointment at McMaster, Dr. Rosenbaum was at the Hospital for Sick Children in Toronto and the Institute of Child Health in London. He is a Fellow of the Royal College of Physicians and Surgeons of Canada.

CHAIRMAN'S APPRAISAL

Chairman's Appraisal

It is the tradition of the Royal Society of Canada that the Chairman appraise and summarize what an inquiry has achieved, and present in consolidated form the conclusions and recommendations. Our recommendations follow immediately. The conclusions are presented afterwards in sections where numbers and titles identify the relevant sections of the main report.

The Commission finds that lead is a very useful heavy metal, largely isolated in natural settings, but now widely disseminated as the result of human economic activity. **We cannot at present do without lead as an industrial material--e.g. in lead-acid batteries--and national consumption is slowly increasing. As a result, we carry body burdens of lead far above prehistoric levels.** We need public policies at all levels of government, and practices in private organizations, that will protect us against the consequences--because **lead is a powerful toxin.** This report deals with the scientific evidence bearing on that need--the science behind the policies.

Recommendations

(These recommendations are addressed towards the most concerned parties, but should be seen as a whole in conjunction with the summary and conclusions that follow.)

To Government

1. **Public health and environmental policy should be to reduce blood lead to its lowest possible level.** At present there is little margin in Canada between the range of observed blood lead levels and the levels at which significant biochemical disturbance has been observed (especially among young

children in the inner cities). Existing blood lead levels in the inner cities are still too high.

2. Because the primary route of uptake of lead into the human body is via food, efforts should be made to accelerate the decline in **lead content of food**, especially for children. Standards, guidelines and voluntary procedures now applied to domestic produce should also be applied to imported goods, especially when the latter are canned.

3. All levels of government should treat lead in the context of overall metal production and consumption, as its use always involves other metals, and vice versa. In particular, **in environmental regulation and monitoring**, attention should be given to **emissions of lead and other heavy metals**:

- by primary producers of iron and steel
- by ferrous metal foundries (especially those treating automotive scrap)
- by the non-ferrous metal industry
- by the lead producers themselves, especially smelters and refiners

Attention should be given to on-site disposal of lead-bearing wastes because these may pose problems in decommissioning the site. Deposition and emission rates, as well as air and water concentrations, should be monitored, and used in guidelines and standards as far as possible.

4. As a matter of urgency, federal and provincial governments should consider **the position of the secondary lead smelting industry**, which is in serious difficulties in Canada and the US. In Canada this industry recycles

lead from 15 000 spent batteries each day. If the industry closes down, the country will need to dispose of these batteries (and other lead wastes) on public sites, and at public expense. Sixty per cent of US capacity is now closed, and some spent batteries are exported to Canada. The Canadian industry urgently needs modernization. Out-of-date facilities lead to contamination of surrounding residential areas.

5. **We endorse the decision of the Government of Canada to eliminate lead from gasoline** from December 31, 1992. We urge a speedy decision as to the means whereby vehicles currently needing lead for valve lubrication may remain in service when leaded gasoline ceases to be sold.

6. We recommend that **MMT** (methyl cyclopentadienyl manganese tricarbonyl) and **MTBE** (methyl tertiary butyl ether) be regarded as environmentally acceptable substitutes for TEL (tetraethyl lead) as octane improvers in gasoline. The same applies to **methanol** and **ethanol** used as additives or blending components.

7. If more severe reforming and isomerization are adopted by refiners as octane improvers (as seems certain), an increase in aromatic hydrocarbons is likely in gasoline. The Government of Canada should examine **the health effects of benzene in gasoline**, and consider establishing limits on the allowable concentration.

8. All levels of government concerned with **drinking water supplies** should be concerned with these points:

- in regions where surface and ground waters are poorly buffered, and precipitation is acidic, water supplies not treated in municipal plants should be checked for lead

- where roof-top cisterns are in use for drinking water, and where precipitation is acidic or contaminated with lead, supplies should be checked for lead

9. The Government of Canada should consider means of warning the public of **the health hazards of paint stripping, scraping or sand-blasting**, which may create lead-enriched dust, especially indoors, where it may be absorbed by children.

10. **Stripping of paint and other surface coverings on large structures in populated areas**, such as steel bridges, should be carried out by wet sand-blasting, or by other dust-suppressing techniques.

11. Provincial and municipal governments should consider **surveys of old paint surfaces** in homes and institutions, and formulate guidelines for removal of deteriorating lead-containing materials. The Government of Canada should consider assisting in the provision of mobile monitoring equipment that might be lent to local authorities for this purpose. There is a serious lack of information about old leaded paint in Canada.

12. Though **lead chromate paint** seems to pose little health hazard, its widespread use on highways calls for caution in application and disposal.

13. **Decommissioning of closed industrial plants, contaminated soil removal and site maintenance** are essential to the prevention of lead contamination. Firm guidelines or regulations are required to control such measures. In particular:

- operators of existing industrial plants with lead-contaminated sites should be required, before ceasing operations, to file formal decommissioning plans with municipal and provincial authorities, including estimates of cost and means of financing
- governments should consider the financial implications of site abandonment by companies lacking the resources to clean up their sites
- provincial governments should compile inventories of sites abandoned in the past, and consider measures for the clean-up of these sites before their use is sanctioned for residential or recreational purposes

14. **Removal of lead-contaminated soils or other surface materials** should be undertaken from the following sites:

- former lead-processing, lead-using or lead-accumulating industrial plants, where soil lead levels exceed those specified in Recommendation 16
- residential, recreational or institutional land (or land to be converted to these uses) contaminated by lead above the levels specified in Recommendation 16

15. **Guidelines governing such removal** should cover the following points:

- each case should deal with the specific conditions applicable to each site (i.e. in a **site-specific** fashion)
- in the case of **soil removal from hot spots** surrounding specific industrial sites, there should be evidence of significant elevation (in the order of 3 μg/dL) of blood lead levels among children of 6 years of age or younger above those typical of urban areas elsewhere
- the **disposal site** should be specified, and be environmentally acceptable
- the **replacement soils** (or other materials) should have lead concentrations and acidity below specified levels

16. In the **decommissioning of old industrial plants and in cleaning up of contamination around existing plants**, the following conditions should apply:

- soil lead levels of up to 500 μg/g are acceptable for residential areas, and for gardens and allotments
- levels up to 1000 μg/g should be acceptable for parklands, and other areas to which children may have intermittent access
- soil removal, if necessary, should be carried out to a depth of 30 cm

Experience suggests that these measures may be ineffective if current deposition rates are high (as they are likely to be until the new regulations on lead in gasoline take effect late in 1987).

17. Adequate guidelines or regulations need to be adopted (where not in place already) concerning **the application of sewage sludge** to agricultural land. The use of untested sludge (which often contains significant lead concentrations) should be discouraged in private gardens and allotments.

18. All levels of government should extend **all possible assistance to community groups in preparing and disseminating educational materials** within hot-spot areas, and among vulnerable groups. There is a great need to raise awareness of the issue, especially as regards personal hygiene, food preparation, diet, care of premises and children's play habits.

To Industry

(Many of the above recommendations require action by industry. In addition, the following specific recommendations are presented.)

19. Primary and secondary lead smelters and refiners should move as quickly as conditions permit **to more modern forms of operation that reduce environmental releases**, and minimize occupational exposures (which, in our view, are still too high, given the available medical evidence).

20. Industry should recognize the need to **involve community groups, labour unions and other work-force organizations** in programmes of clean-up, soil removal, environmental control, health monitoring and awareness of lead toxicity.

To Research Laboratories and Community Groups

21. Investigators should consider the **use of lower plants as biomonitors** in determining the time trend of lead deposition, especially during the period of rapid lead phase-down in gasoline.

22. Research is needed into the **risk of human exposure** at locations where soil lead levels are elevated. This should include further investigation of the available models relating soil lead concentrations to uptake and blood lead levels, determination of the significance of chemical and physical forms of lead in soil, and assessment of the age of the deposited lead.

23. **Methods of sampling soil** should be standardized so as to yield representative and mutually comparable levels of soil lead.

24. In hot-spot areas, **lead levels in vegetables and fruit** from urban gardens (including highway borders) should be checked. Washing, removal of outer leaves and peeling of root vegetables are useful precautions.

25. **Fish and seafood from contaminated or suspect water bodies** should be tested for lead.

26. The relationship between **lead in acidic versus neutral water**, and uptake by fish, also needs investigation. Lead is not the only metal that may accumulate; cadmium and mercury also accumulate in fish from acidic lakes.

27. Further research is needed into the occurrence and **biological transformations of organolead** in aquatic ecosystems.

28. Because **the best measure of exposure to lead is blood lead level**, it is essential to ensure comparability of results. When surveys are conducted, whether in adult or in child populations, strict, well-standardized and control routines for blood collection and storage should be adopted. Venous blood only should be used (for children and adults alike) unless experienced persons are available who have been specifically trained for pinprick blood collection. For inter-survey comparisons, venous blood levels are essential.

29. **Surveys of blood lead levels** should always be undertaken in the identification of hot spots, and should be conducted annually during periods of clean-up. Wherever possible, such surveys should also be included in surveys undertaken for other purposes.

In General

30. All the above recommendations, to be effective, require the prompt co-operative actions of all the responsible agencies in all levels of government, and of all other parties, inside and outside government. The obstacles to further reduction of lead exposure are institutional, not technical.

Summary and Conclusions

The summary that follows includes the Commission's conclusions on which the above recommendations are based. The headings correspond to the numbered sections in the report.

I How Serious an Issue is Lead?

1. Most members of the public seem little concerned with this issue. Our advertised public meetings attracted little interest. Many drivers systematically alter their cars so that they can use leaded gasoline (misfuelling), disregarding any threat to their own health and that of others. Such misfuelling inactivates catalytic converters. Even in urban hot spots most residents seem uninterested.

2. Hospital emergency department separations (i.e. diagnoses on discharge) show that lead poisoning is a rare event among Canadian children, and chelation therapy is rarely necessary in Canada. It is insignificant compared with poisoning by aspirin or other drugs. Accidents are an overwhelmingly greater danger than lead or drugs. Few clinicians have much experience with lead poisoning.

3. There may, however, be more subtle effects arising among children, and perhaps others, at the low lead exposures typical of Canada. These are the main concern of the report and the main reason why further measures are needed, even though conditions have been rapidly improving.

II Present Status of Lead in the Canadian Environment

1. It is difficult to monitor lead concentrations in the environment. In particular, it is costly to derive representative values for water and soil, and to monitor their change with time.

2. Ambient air concentrations in Canada have been falling steadily since 1973, and are currently well below those thought to be typical of some other industrialized countries. They should fall still lower when lead is further reduced in gasoline, starting in 1987.

3. In the US, a similar fall (though starting at levels above Canada's) has almost exactly matched the fall in gasoline lead emissions. This fit is also good in Canada. However, ambient air lead concentrations have also fallen in several countries where leaded gasoline has remained in widespread use.

4. It is not possible to say whether similar improvements have occurred in soils and water bodies. Such improvement seems unlikely, because lead accumulates (and is thereafter virtually immobile) in soils and sediments.

III Lead Industries

1. Canada is the western world's third largest producer of mined lead, fifth largest producer of refined lead and second largest exporter of lead concentrates and refined metal. Since 1979, prices have been very low, and the industry has had to struggle. In the mining and milling sector, Canada is competitive, but her smelters and refiners have one of the world's highest cost structures.

2. Canadian lead consumption has been growing slowly, but is much lower than national production (hence our exports). The main export markets have been the US, Europe and Japan. All are expected to remain highly competitive for the foreseeable future. Primary lead producers (now mainly two, Cominco Ltd. and Noranda Inc., through Brunswick Mining and Smelting Corporation) hence operate in a difficult price environment. Their operations require expensive and elaborate environmental controls.

3. 1982 estimates (the most recent inventory) are that 73 000 tonnes of lead were discharged into the Canadian environment. Of this amount, 61 000 tonnes were solid wastes, largely inert. The vital part was the 11 446 tonnes emitted in particulate form to the atmosphere (7278 t, or 63% of total emissions, from leaded gasoline). The rest of the emissions came chiefly from mining, smelting and refining plants (including considerable amounts from iron, steel and non-ferrous metal production). Effluents to water were only 562 t, most of it (412 t) in sewage sludge (from gasoline and industrial lead carried via the sewers). Solid wastes added directly to soils were large in bulk, but low in chemical activity (except for gasoline-derived particles, especially near highways).

4. Lead minerals occur in close association with those of zinc, and the ores contain several other heavy metals in low concentrations. This means that:

- zinc production requires by-product lead, usually as a concentrate; hence lead output cannot be curtailed or expanded simply in response to price changes and demand for lead itself

- wastes from lead production (or that of zinc) always contain other heavy metals, some of which (especially cadmium) are highly toxic
- lead regulation and monitoring always ought to be seen in the context of overall protection against the heavy metals as a group

5. The secondary lead smelters, of which there are at present eight in operation, are essential to overall operation of the industry and to environmental clean-up. They recycle lead wastes, most notably 15 000 expired lead-acid batteries per day. Recycling efficiency is said to be 98%. Without the secondary smelters these wastes would be dumped, and might become a serious hazard. Some jurisdictions outside Canada offer consumer incentives to encourage proper disposal of batteries. The secondary lead smelters have special responsibilities for environmental protection, because they operate so close to people.

6. The secondary smelters include three in urban areas near housing, and have long created environmental problems because of lead escapes, though this situation has improved. The industry is now barely profitable. In the US, recycling capacity has fallen from 1.1 x 10^6 t of lead capacity per annum to a present operating level of 400 000 tonnes. By 1990 nearly 1.4 x 10^6 t of expired battery lead may have been dumped in the US (and some exported to Canada, as at present). If secondary smelting of lead and the transportation of used batteries were to be regulated under the Canadian Hazardous Products or Transportation of Dangerous Goods Acts, the industry might be forced out of business. This would result in the same dumping problem as that now being experienced in the US.

7. The major lead-using industries in Canada are the manufacture of lead-acid batteries, paints and coatings (some now considerably reduced), various chemicals, solders, lead-soldered cans, telephone and power cables, and a variety of uses in piping, roofing and electrical equipment. The use of lead paint (now confined mainly to exterior surfaces) continues to pose occupational exposures, and may occasionally affect others--chiefly when old paint flakes or is sand-blasted. This does not seem to be as bad a problem as it appears to be in the US. Other forms of lead use are adequately regulated, and do not pose serious environmental hazards.

8. The low profit levels (if any) in primary and secondary lead smelting and refining have delayed plans to modernize equipment. Some of the environmental failures in these industries arise from the use of out-dated technology.

IV Use of Lead in Gasoline

1. Tetraethyl lead (TEL) has been the principal octane-improving additive in Canadian gasoline since 1926. Its use began to be phased down in 1974, when a federal standard of 0.77 g/L for lead in leaded gasoline was imposed. Consumption of lead in TEL led to emissions of 14 360 t in 1973, the peak year. They have now fallen below 7000 t. They will fall again to 2750 t as the result of new regulations in 1987, and to 141 t after further restrictions in 1992. Thus, the major source of lead in dust and soil is being rapidly removed.

2. The octane deficit incurred by the withdrawal of TEL will have to be filled by other additives or by altered processing technology (a reduction in octane requirement is unrealistic in the face of engine performance requirements). Both are likely to be used by refiners, as they shift out of leaded gasoline.

3. Of the additives, MMT (methyl cyclopentadienyl manganese tricarbonyl) is at present controlled by the Canadian General Standards Board at 0.018 g/L. Manganese is an abundant element in nature, and we are exposed to it at levels far exceeding those that will arise from its use as a substitute for TEL.

4. Increased use of methanol and ethanol also presents no predictable health hazard, though there are technical problems to be overcome when these fuels are used as additives.

5. MTBE (methyl tertiary butyl ether) is an attractive blending component for raising octane. It is blended up to a maximum of 11% of gasoline in the US. There are no manufacturing facilities in this country. Raw materials required are methanol (of which Canada has a surplus) and isobutylene, which has to be imported (though we could produce it). There are no known health implications.

6. The remaining demand for octane will be met by capital investment to increase the severity of reforming, and by other changes in the refining processes, including isomerization. The capital cost of the entire phase-down from 0.45 g/L (the present actual level) is estimated by the industry to be

$800 million for a lead standard of 0.026 g/L after 1990. It is our opinion that the industry is likely to achieve its objective at a lower figure, because (i) it has negotiated a later date for introduction of the new standard (expected to be January 1, 1993), and (ii) considerable technological advances are being made by US refiners, who have had to meet this standard much sooner. We estimate an increase in the retail cost (for the total phase-down) of gasoline not exceeding 2.1 cents per litre to meet this target (in the 1985 sales/tax regime).

7. A consequence of these refining or additive changes will be an increased concentration in gasoline of certain aromatic hydrocarbons, chiefly benzene, toluene and xylene. Some of these hydrocarbons are toxic, and others (notably benzene) are carcinogenic in both animals and humans. Hence we think it prudent to minimize the presence of aromatic hydrocarbons in gasoline.

V Environmental Pathways of Lead

1. Atmospheric emissions of lead from the human economy, on a global scale, are the prime source of lead moving through or stored in the human environment. Living organisms (biota) absorb some of this lead from soil or water, but these reservoirs receive most of their supply from atmospheric fallout.

2. The residence time of lead particles in air is quite short (10 d on the average). Hence, deposition (air fallout) rates are a better measure of the lead burden in the environment than is the usual measure (ambient concentrations in air).

3. Lead is dispersed to remote areas by the winds, as evidenced by lead in glacial ice, lake sediments and plant monitors. Lead in mosses and lichens collected from natural communities reflects lead deposition.

4. Lead in soil is persistent, and can be a source of uptake by plants, animals and young children.

5. Vegetables and low-growing fruit (e.g. strawberries) can be contaminated by lead from air by direct deposition, or from the soil. Only lead from air affects taller fruit. Soil splash containing lead is likely to be more significant for plants than is transport from the soil via their roots.

6. Lead in the environment is not a threat to biota in terrestrial or aquatic systems, except for those close to point sources (such as mines, smelters and highways), or in other regions of high contamination. Human targets are less tolerant.

7. Toxicity of lead depends upon its chemical form, as well as other physical and chemical factors, and the inherent sensitivity or tolerance of the organism. Organic lead (such as TEL) is much more toxic than inorganic lead (such as lead ore dusts).

8. Lead accumulates and persists in sediments, where it can be taken up by sediment-dwelling (benthic) organisms. It may then be passed along the food-chain.

9. Lead is not biomagnified through the food-chain, but instead tends to decrease in concentration at successive trophic levels. This has been demonstrated repeatedly for aquatic systems, both freshwater and marine, and less frequently for terrestrial systems.

10. Polluted precipitation contributes lead to the terrestrial and aquatic environments, as evidenced by the increased concentration of lead in precipitation over regions receiving acidic deposition. The significance of this addition of acidic material in terms of demonstrated or potential effects on human health is unknown.

11. Lead already present in the environment may be mobilized from soil, sediments or vegetation by acidic deposition, as a result of increased solubility. Generally, this occurs only when the pH (level of acidity) is below 4.0, and there is no confirmation that this actually happens in the environment.

13. Lead in drinking water may be elevated in regions where water is soft and acidified, but in most cases the lead source is the delivery system. The leaded water can be flushed out by allowing the tap to run after a period of standing. We have not found evidence that this is a significant problem in Canada.

13. Roof-top cisterns for collection of domestic water have been shown to result in unacceptable lead levels in drinking water. The lead in these instances cannot be removed by allowing the tap to run.

14. Acidified ground water or surface water used directly for domestic supplies may contain unacceptable lead levels in regions where rocks are siliceous and insoluble, and water is soft. Running the tap will not remedy this problem. Again, we have not encountered this problem, but suspect it may exist locally.

15. Fish in acidified lakes have been shown to contain more lead than do fish in more neutral lakes in otherwise comparable environments. This relationship is statistical, but is supported by experimental evidence. Lead in fish accumulates mostly in kidney, liver and bone, rather than in the muscle tissue that is normally consumed by humans.

VI Uptake of Lead by Humans

1. There are two major routes of uptake (absorption of lead) for human beings: inhalation (from air) and ingestion (from water, beverages, dust and soil). Intake is the total entering the body, uptake the total retained.

2. Blood lead is related to the rate of uptake of lead. Data from several researchers agree that blood lead (μg/dL) is approximately 40% of daily intake (μg/d) at equilibrium, even though children and adults differ in rate of uptake.

3. For inhalation, the uptake of inhaled lead is approximately 50% of the total amount inhaled for submicrometre aerosol particles. Deposition in the respiratory system depends upon particle size; larger particles do not usually reach the deeper parts of the respiratory tract, and for them uptake is much less than 50% of total inhaled lead.

4. For inorganic lead, particle size is more important than the chemical form of lead in determining the uptake/intake ratio.

5. For ingestion, the ratio of uptake to intake is much more variable than for inhalation, ranging from 80% to less than 10%. The amount and type of food, nutritional status, age and sex all affect the ratio. Generally, children appear to absorb more lead from their diet than do adults. A conservatively safe overestimate of the ratio of uptake to total intake is 15%.

6. Food and beverages used to be severely contaminated from solder in cans, but this source has been largely eliminated. However, young children may still absorb three times as much lead per unit body weight as do adults.

7. Lead in food includes lead taken up by plants, which is rarely a major source except for (possibly) certain leafy vegetables grown or stored in areas of high lead deposition.

8. Lead can be elevated in certain fish or shellfish collected from contaminated environments, even in the absence of toxic effects on the fish. This can be a dietary source of lead for people. Fish and shellfish catches may have lead concentrations exceeding the guidelines for human consumption.

9. Lead in soil may be ingested, especially by young children, because of handling and eating of soil, mouthing and dust contamination of domestic objects. But the quantitative significance of this route is still controversial. Populations living in areas with highly contaminated soil do not always show corresponding elevation of blood lead levels. A recent estimate of blood

lead/soil lead from England is 0.6 µg/dL per 1000 µg/g of lead in soil. There is considerable disparity among studies of this relationship, which is of great potential significance in the clean-up of contaminated soils. Intake depends primarily on recent additions to soil lead.

10. Methods for sampling lead in soil are quite variable, and care is needed, especially when comparing the results of different studies, to determine exactly what is meant by lead in soil. Is it from surface grabs, scrapings or integrated cores?

11. Quantitative models can be useful in estimating exposure to lead from various sources, but there are considerable discrepancies among different studies, because of uncertainties in intake, uptake and source/blood lead transfer. Models can nevertheless provide information influencing decisions on regulatory levels and control strategies.

VII Health Effects of Lead Exposure

1. Blood lead levels in Canada--the most acceptable measure of recent exposure to lead--are at the lowest levels of concentration ranges reported for all countries from which comparable data could be obtained. Canada is not free of the problem, but seems better off than many others.

2. This relatively favourable situation is true even in the hot spots reported in Section VIII. The proportion of children with blood lead above the recommended intervention level of 25 µg/dL is substantially less than in large urbanized areas of other countries.

3. We believe that this advantage arises mainly from the recency of growth of Canadian cities; from the rarity of inner-city dilapidation; from high housing and public health standards; and from the vigilance of civic authorities and neighbourhood groups. However, blood lead levels in the inner cities, especially among children, are still not matters for congratulation. They should be lower.

4. Certain Canadians are more at risk than others, because of differences in the distribution of lead in the environment; differences in personal habits and hygiene; and differences in rates of absorption and forms of metabolism. Women, especially when pregnant, and young children are the most vulnerable groups, as are lower-income groups generally.

5. Lead is a multi-system toxin; it is known to have adverse effects on several systems in the human body. Nevertheless, the number of cases of overt (frank) lead poisoning in Canada is very small.

6. There is no deterministic or categorical way in which we can establish a point at which lead can be said to have harmed an individual at low levels of exposure.

7. Heme biosynthesis and other metabolic processes are affected. Lead inhibition of the enzyme ALA-D can be noted at blood lead levels as low as 10 µg/dL. Heme biosynthesis is probably not decreased until the ALA-D concentration falls below 20-30% of normal. Ferrochelatase inhibition is established at levels above 35-40 µg/dL, but anemia does not usually appear until levels

reach 50 µg/dL in adults, and 40 µg/dL in children. Erythrocyte protoporphyrin (EP), a product of these changes, appears to increase exponentially above a level of 15-18 µg/dL in children, and above 35-40 µg/dL in adults. 1,25-dihydroxy vitamin D is depressed at blood levels above 33 µg/dL.

8. Much controversy persists concerning the nature of the relationship between blood lead and neuropsychological effects in children. Two major problems are:

- the difficulty in determining the relative contributions of lead and socio-demographic factors to neuropsychological functions of children
- the difficulties in interpretation arising from the smallness of the effects commonly attributed to lead at the blood lead levels encountered among children. Current studies being conducted prospectively have yet to confirm the observations reported in earlier studies

However, the consistency of the direction of the association, even after statistical adjustment for confounding variables, suggests that the effect is indeed real, though small.

9. Other effects on the central nervous system have been suggested by neurophysiological studies of the central nervous system in children. These studies have yielded conflicting results that are hard to interpret. The Commission considers them to be still experimental.

10. Effects on the peripheral nervous system have been examined. There is little doubt that nerve conduction becomes altered at blood lead levels as low as 50 µg/dL, but evidence of change at lower levels is inconclusive.

11. An association between blood lead levels and blood pressure was substantiated in certain groups in the US NHANES II study. A similar weak association was found in one industrial cohort in Canada. A causal connection between blood lead and blood pressure remains to be established. In any case, the association, if real, is small. The available evidence is insufficient to yield assumptions on which to base estimates of savings in health costs that might arise in Canada from reductions in blood lead levels in the adult population.

12. The Commission does not believe that major surveys dedicated to assessing lead burden in the Canadian population need to be undertaken. Surveys made for other purposes should, however, include measurements of blood lead. Such surveys should be more widely undertaken in the identified hot spots. Soil removal should be undertaken only in those areas where blood lead levels in children are significantly raised.

13. The great volume of research conducted in the past 5 years, chiefly outside Canada, has not demonstrated that serious health effects exist among the Canadian population, which is exposed on average to less lead than are the people of other industrialized countries. Clinical experience suggests that serious cases of lead poisoning are very rare, and has not detected the more subtle effects discussed above, notably those concerned with heme synthesis.

14. Nevertheless, the Commission repeats a conclusion it reached in its interim report: the effects of low levels of blood lead on the functioning of the hematopoietic system (notably on heme synthesis) justify reducing blood lead in the population below 1986 levels. The case for doing so rests on prudence rather than on clearly demonstrated harm; there is very little gap between observed blood lead levels and levels at which significant biochemical disturbance is observed.

VIII Hot Spots

1. Local areas of potentially higher lead exposure, called hot spots, are located in two chief kinds of site:

- near secondary lead smelters or metalworking plants in urban areas; and possibly near ferrous foundries using automotive scrap
- near primary lead smelters, mines or concentrators; near primary iron, steel and non-ferrous metal-producing installations

2. Only a few of these potential sites have drawn public attention: notable examples are South Riverdale and the Niagara Neighbourhood in Toronto; the Weston School area of Winnipeg; and the primary lead producers at Trail and Kimberley, BC, and Belledune-Bathurst-Dalhousie, NB. There are probably other areas of contaminated soil across the country where former lead-working facilities have been abandoned, often long ago. One such case was recently discovered within a major Ontario city.

3. In urban hot spots there are usually elevated levels of soil lead near the plant, and dust may also contain much lead. Blood lead levels that

are substantially above ordinary urban levels have been observed among children, though strong action by neighbourhood groups, local health authorities and the companies concerned have sharply reduced lead emissions in the past decade. It should be a fundamental principle that no child should suffer a significant penalty in body lead burden because of where he or she lives. Blood lead levels among children in the Toronto hot spots have returned to near normal for urban exposures, except in one area where they continue about 3 μg/dL above general levels. Few children now appear to have venous blood levels above 20 μg/dL (though information is very scarce).

4. Gasoline lead continues to contribute to the total uptake of lead in all urban areas, and reduction of smelter emissions will not alone reduce blood lead levels to normal values. Soil lead and flaking paint may also contribute, though the latter has not been shown to be a significant source of lead for children in this country.

5. The primary mining, smelting and refinery areas at Trail-Kimberley and Belledune-Bathurst-Dalhousie are remote from large concentrations of people, and do not appear to threaten local residents. Emission regulations appear generally to be met, though at Trail the plant operates under a ministerial variance of the original provincial order. Effluent controls now appear effective, after failures. Earlier high exposures in Trail led to elevated blood lead levels among children. Though there has been no recent survey, these appear to have fallen. Further environmental improvements in both areas will have to wait for modernization of the production facilities. There is much anxiety in these communities, not about lead exposure, but about jobs and the future of the industry.

6. Leaded paint does not seem to be much of a problem in Canada. We encountered no case where it was definitely implicated in raised blood lead levels among groups of children, though this has since been suggested as a possible cause in the case of the highest levels in South Riverdale. There is, however, a general lack of information about old leaded paint, and this needs to be rectified.

7. There is also a lack of awareness of the potential hazards involved in the removal of old leaded paint. There is a need to advertise this hazard more widely.

8. Canada needs a more thorough and standardized approach towards the decommissioning and decontamination of old industrial sites, especially those impregnated by lead wastes. Where these are in areas likely to be used for housing or cultivation, adequate guidelines (and perhaps standards) are needed for renewal of soils. Soil removal in existing contaminated housing areas also needs adequate controls. It would be wise to defer soil removal until after the next acceleration in the phase-down of lead in gasoline (i.e. to the summer of 1988); otherwise the site may be contaminated again.

9. The same applies to the use of sewage sludges on agricultural land. Much of the waste lead from urban areas escapes in such sludge. Firm guidelines are needed to control its use in cropped areas (where, however, its other properties may be very desirable). Use of untested sludge in gardens and allotments is not advisable.

10. In reducing hot spots to levels of lead exposure typical of other urban areas, a key element is education. Personal hygiene, diet and play habits play major roles in determining uptake. Local neighbourhood groups, community organizations and national public interest groups all have important roles to play in this process of education. They can also help in monitoring performance, and in drawing public attention to new hazards or failures in technology. Co-operation between such groups, local health and environmental authorities and companies is growing, but not fast enough. It should be encouraged by all possible means.

IX What does the Situation Imply for Canada?

1. There will be a continuing market for lead within Canada (and for Canadian exports) for many years. Both markets will, however, remain highly competitive. It will hence be difficult to modernize facilities.

2. Canada needs to see the lead industry as a whole, and to consider its waste, recycling and environmental problems as part of that whole. The jurisdictional tangle surrounding the industry is an obstacle to such an integrated view.

3. Nevertheless, Canada can well afford to rid itself of the remaining hazards posed by the use of lead, and should do so.

Acronyms/Abbreviations

Because we constantly refer to various reports, agencies, substances and terminologies in the text, we have used the following acronyms and abbreviations:

AAACLD: Alberta Association for Adults and Children with Learning Disabilities

ALA : delta-aminolevulinic acid

ALA-D : delta-aminolevulinic acid dehydratase

ALA-S : delta-aminolevulinic acid synthetase

AR : accumulation rate

CEC : Commission of the European Communities

CF : concentration factor

CMA : Central Metropolitan Areas

CMVA : Canadian Motor Vehicle Manufacturers' Association

CP : coproporphyrin

CSA : Canadian Standards Association

DRI : Data Resources of Canada

EEC : European Economic Community

EF : enrichment factor

EMR : Energy, Mines and Resources Canada

EP : erythrocyte protoporphyrin

EPA : The United States Environmental Protection Agency

ESSA : Environmental and Social Systems Analysts Ltd.

FAO : Food and Agriculture Organization

FCC : fluid catalytic cracking

HWC	Health and Welfare Canada
HWR	Report of the Commission's Health Workshop, "Health Effects of Lead"
ILZRO	International Lead Zinc Research Organization
LDAC	Learning Disabilities Association of Canada
MMD	mass median diameter
MMT	methyl cyclopentadienyl manganese tricarbonyl
MTBE	methyl tertiary butyl ether
NAPS	National Air Pollution Surveillance
NHANES	National Health and Nutrition Examination Survey
PACE	Petroleum Association for the Conservation of the Canadian Environment
Pb	Lead
PbA	Lead in air
PbB	Lead in blood
PbD	Lead in dust
PbF	Lead fallout from atmosphere
PbS	Lead in soil (also denotes lead sulphide)
PbW	Lead in water
PCB	polychlorinated biphenyl
pE	redox potential
pH	negative logarithm (to base 10) of hydrogen ion concentration, a measure of acidity
PVC	polyvinyl chloride
PWR	Report of the Commission's Pathways Workshop, "Pathways, Cycling and Transformation of Lead in the Environment"
RSC	Royal Society of Canada
TEL	tetraethyl lead

TML : tetramethyl lead

UN : United Nations

VER : visual evoked responses

WHO : World Health Organization

THE MAIN REPORT

I Preamble

1. The Issue

Lead is a very useful heavy metal. In nature it is segregated, chiefly buried in insoluble and biologically inoffensive forms in the environment. However, human beings have disinterred it and, in so doing, released large quantities into the air, soils and surface waters. From there it has entered the human body.

Each of us now carries a substantial burden of lead, whereas our prehistoric ancestors had only traces in their tissues. Because lead is very toxic, this body burden is a potential threat to health. Large doses of lead produce sickness or even death. Smaller doses affect various systems within the body. Even the small burdens typical of the Canadian population have been thought dangerous. We have lived with these hazards for centuries, but have recently become sensitized to their implications, especially for our children and pregnant women. Is their well-being at risk?

Certainly some Canadians believe so. The most insistent intervenor in the Commission's affairs has been the Learning Disabilities Association of Canada (LDAC). In its brief to us urging the removal of lead from all possible sources, its spokeswoman, Mrs. Barbara McElgunn, wrote: "We cannot afford morally or economically to take risks with the health and ability of Canadian children." Her words expressed an anxiety shared by a number of those who approached us.

The Commission has seen its job, however, as not just to assess the health hazard of lead, though we have carefully examined this subject. Lead is so useful a metal--e.g. in lead-acid batteries--that we cannot do without it until technology advances radically. If we minimize the use of lead, as public interest groups urge, we put the jobs and capital investment required by the industry in jeopardy, and also change Canada's balance of international payments. These sacrifices may be necessary, but they will certainly be expensive for some. We have hence looked at the economic consequences of lead phase-down.

We have not attempted to write an encyclopaedia about lead. There are several excellent overviews of the metal as an environmental and health factor. A noteworthy effort is the National Research Council of Canada's useful document, "Effects of Lead in the Environment - 1978. Quantitative Aspects" (Jaworski, 1978). Also excellent are the massive criteria documents of the US Environmental Protection Agency (EPA, 1986) and the ninth report of the UK Royal Commission on Environmental Pollution, "Lead in the Environment" (Southwood, 1983). We have tried to build on these impressive foundations, and to apply our understanding to the specific problems of Canada.

This report begins with an analysis of sources of environmental lead--overwhelmingly, as far as chemically active forms are concerned, derived from the human economy. It is our material culture that has remobilized this toxin, and exposed our bodies to its effects. We look at the mining of lead, its metallurgical and chemical transformations and its use within the economy. We describe some of these processes in technical detail, in the belief that environmental regulation and control require such understanding.

We next examine the environmental pathways whereby lead can reach specific human targets. As far as we can judge, the environment itself--air, water, soil, plants and animals--is not harmed very much by the lead released by human activities, but it is certainly altered. And these changes affect the way in which human beings are reached by lead. Most of us get the lead in our tissues from the food we eat, and secondarily from the water we drink and the air we breathe. The functioning environment delivers these doses. So we have tried to analyse the pathways travelled by lead. How does it reach us?

After the lead has reached us, how do we take it in and absorb it? Once within our bodies, what does it do to our blood, nerves, other tissues, health and bodily performance? Is it the massive public health problem that it seems to be in some US cities? Is it, in fact, a burden that our children should not be required to bear?

Political attention has been focussed in recent years on the private car and the leaded gasoline that it spreads throughout the world, not least on the streets of Canadian cities. We have examined this issue, and in our interim report ("Lead in Gasoline: A Review of the Canadian Policy Issue") advised the Canadian government on measures it might take to reduce the problem. Action is being taken along lines consistent with our advice. In the present report we consider future conditions, and also review the technological issues involved in preserving engine performance in the absence of leaded gasoline.

Are further measures required to protect the Canadian public? In this final report we offer, as briefly as we can, an overview of the problem, and answer the question. Our expertise is scientific, medical and technological, as is the answer we give. We have pointed to the jurisdictional, legal and administrative implications of our response, but have not specified how matters should be handled. There are many people within federal and provincial agencies who are far more expert than the Commission in such matters.

We conclude that further measures are indeed needed. Overall, however, the situation is rapidly improving, thanks to actions already taken by individuals, governments and corporations. For once, an environmental appraisal can sound an optimistic note.

2. How Serious an Issue Is Lead?

Three pieces of evidence suggest to the Commission that most Canadians are little concerned about lead in the environment:

- the fact that our own public meetings were poorly attended, in spite of wide advertising
- the fact that we were offered little evidence that actual harm was being done to members of our society
- the widespread occurrence of misfuelling, the practice whereby consumers adapt their cars (or the nozzles of gas pumps) so as to use leaded gasoline in cars manufactured for unleaded gasoline (evidently many drivers disregard the implied threat to health)

We shall have more to say about these points later. Meanwhile we have to admit that the Commission encountered little interest in its work among the public at large, and actually had to finance an adequate response from certain sectors. The review of our procedures by Margaret C. Grisdale in Appendix I emphasizes this point.

On the other hand, a small number of public interest groups made strong representations to us, especially as regards the perceived threat to child health arising from the continued use of leaded gasoline. Several of these groups represented residents of central-city neighbourhoods near secondary lead smelters, and had originally come into being because of overspills of lead into the surrounding urban environment. We also received representations from national organizations, notably Friends of the Earth, the Canadian Environmental Law Association and the LDAC. All the groups favoured further action, and told us that in their view lead was a continued threat to child welfare.

There is evidence that lead poisoning is a much less common hazard for children than are traffic accidents, drug misuse and accidents in general. Table I.I shows the comparative statistics for hospital emergency department separations (diagnosis on discharge). In Appendix VI we offer an alternative analysis of hospital statistics, separating the responses by age group. Both analyses confirm the rarity with which lead now presents itself as a cause of acute illness.

No Canadian clinician to whom we spoke had much experience with lead poisoning in children, in striking contrast to certain US physicians. Some large US hospitals have special lead poisoning clinics (e.g. the Montefiore Medical Center in New York). We wrote to the senior health officials in all cities, asking for their advice. We convened a meeting of the Directors of health services of the Maritime Provinces (where paint flaking, if a problem, might have been detected). In nearly all cases we received the same reply: this was not a matter of particular concern within their jurisdictions, and they themselves did not see it as critical. One exception was Toronto, where the Medical Officer of Health and the Board of Health had indeed dealt with anxieties raised by conditions around lead smelters. However, even here there was no evidence of serious harm to children, and related hospital admissions had fallen to very low levels (these were all concerned with accidental exposures, not chronic environmental exposures). The

Table I.I

Comparison of Number of Hospital Emergency Department Separations* for Various Causes of Poisoning and for Traffic Accidents, for Children Aged under 1, 1-6 and 7-14 Years, Canada, 1979-1984

	1979-1980			1980-1981			1981-1982			1982-1983			1983-1984		
Classification	< 1	1-6	7-14	< 1	1-6	7-14	< 1	1-6	7-14	< 1	1-6	7-14	< 1	1-6	7-14
By nature of injury															
Inorganic Pb	0	0	0	0	0	0	0	3	0	0	1	0	2	1	1
Organic Pb	0	0	0	0	0	0	0	0	0	0	1	0	0	0	1
Other Pb compounds	0	0	1	0	0	3	0	0	1	0	0	1	2	1	2
Non-specified Pb compounds	3	1	3	0	1	3	0	3	2	0	0	5	0	0	1
Total Pb	3	1	4	0	1	6	0	6	3	0	2	6	4	2	5
All other metals	0	12	0	1	14	0	2	10	5	2	6	0	3	11	4
Aspirin	12	518	269	16	457	208	17	607	283	14	830	278	12	555	221
All other drugs	163	2817	1153	177	2796	967	162	2760	1207	150	3015	1193	137	2668	956
All non-medicinal household substances	183	1769	413	154	1661	363	136	1456	310	133	1498	293	86	1356	268
By external causes															
Pb and its compounds**	-	-	-	-	-	-	0	1	8	0	4	5	0	3	3
Pb in paints**	-	-	-	-	-	-	0	2	0	0	0	0	2	1	0
Traffic accidents**	-	-	-	-	-	-	126	2218	4139	129	1849	3496	69	1443	2680
Other accidents**	-	-	-	-	-	-	3830	20233	23943	3753	20438	23603	3496	18497	21369

* Hospital "separations" mean, in this table, the diagnosis of cause of injury on discharge.
**Not available for all provinces prior to 1982-1983.

Source: Statistics Canada, 1986

Vancouver Health Department was aware of the threat, but offered no evidence of exposures greater than intervention levels.

Because of stronger public anxiety in the United States, action has already been taken by the EPA to restrict, and then eliminate, the use of lead in gasoline. EPA's actions have been very rapid and drastic, being prompted by growing evidence of serious problems of child health in big US cities, where cases of actual lead poisoning among children continue to be widespread, and where the number of children with blood lead levels above intervention levels is still high.

EPA's decisions rest on a formidable body of new medical evidence, mostly gathered in the past decade. The dramatic response of the US government has been the driving force behind calls for similar action in several other countries, including Canada, the United Kingdom, other countries of the European Economic Community (EEC), Australia and New Zealand. We have been in touch with authorities in all these jurisdictions, and summarize their positions later in the text.

The Commission has received much evidence, and considerable pressure, from the Canadian lead-related industries, as well as from the International Lead Zinc Research Organization (ILZRO). These industries are seriously alarmed at the threat to their markets from the lead phase-down. We also received strong representations from labour unions who see their members' jobs being threatened, and from the mayors and public bodies of several towns with large stakes in the industry (such as Trail and Kimberley, BC). The lead alkyl industry was actually moving towards closure of Canadian production during our work.

We give our detailed conclusions in the Chairman's Appraisal, but state here that we are agreed as follows:

- that most Canadians, like the members of other industrialized urban societies, carry body lead burdens well above those typical of peoples living in remote parts of the world. This alone requires that we be aware of lead's toxicity, and have national policies (federal and provincial) to ensure the protection of all our citizens

- that we are less exposed to this hazard than are most other industrial societies, and that there are few Canadians at serious risk at present. Those Canadians at serious risk are chiefly occupationally exposed individuals (excluded from our Terms of Reference), and persons living in or near certain hot spots where local measures of protection must be taken

- that there are still grounds for disquiet about child diet. A highly qualified authority (Dabeka, HWR [see p. 10 for explanation]) told us that the average Canadian infant is consuming three times more lead per unit body weight than is the average adult. In some cases the average infant may be consuming more dietary lead than is recommended for adults by United Nations (UN) agencies (Food and Agriculture Organization [FAO] and World Health Organization [WHO]) from all sources. Clearly this is an unsatisfactory situation

- that the measures already taken or announced by the Canadian government are or will be sufficient to remove most of the remaining hazard.

The Commission will suggest in its recommendations what remains to be done.

In summary, we find the issue to be real and calling for public action. However, Canada's situation is better than that of other industrial countries, and is currently improving; we believe that few people are now seriously at risk from the presence of lead in the environment. We need measures that will protect those Canadians still threatened.

3. The Commission's Work and Reports

Appendix I, written by the Manager, Margaret C. Grisdale, gives a detailed overview of the Commission's work. Only a summary is given here. We began work in December, 1984, and expect to disband in December, 1986.

We depended on our individual experience and work, and on those of a small professional staff. We established contact with individuals and groups who were concerned with the issue or skilled in the fields involved, not only within Canada, but also in the US, UK and the European Community (CEC) countries. With their help we built up a knowledge of the literature, and of specific investigations under way. We held both public and private meetings with individuals and organizations. Public meetings were held across the country. We remained in close touch with federal and provincial governments, and with public health officials in the major Canadian cities. We also visited many of the areas where lead in the environment had become a prominent issue.

Concluding that we could not rely entirely on our own efforts, we invited and received numerous briefings and documents from government, industry, public interest groups and individuals. We funded certain specific studies.

All these are listed in Appendix II. In addition, we employed numerous consultants, among them DRI (Data Resources of Canada) (in economic matters) and ESSA (Environmental and Social Systems Analysts Ltd.), the latter in assessing urban pathways.

Our reports cover all the Commission's work, plus that of its advisers:

I This final report, "Lead in the Canadian Environment", summarizing our findings and recommendations, dated September, 1986.

II An interim report, submitted in September, 1985, "Lead in Gasoline: A Review of the Canadian Policy Issue."

III A supplementary report, submitted in January, 1986, "Alternatives to Lead in Gasoline", by Marcus C.B. Hotz.

IV The report of our health workshop (held in March, 1985), "Health Effects of Lead", dated September, 1986. This includes technical papers by Commissioners, consultants and external experts (edited by Marcus C.B. Hotz). In subsequent references this document is identified as HWR. (For contributors and titles, see Appendix VIII.)

V The report of our pathways workshop (held in October, 1985), "Pathways, Cycling and Transformation of Lead in the Environment", including technical papers by a further team of experts (edited by Pamela M. Stokes, Vice-chairman). This is referred to in the text as PWR. (For contributors and titles, see Appendix VIII.)

VI Reports prepared under contract by organizations with special interest in the issue. These include reports from the South

Riverdale Community Health Centre (prepared by M.M. Rachlis and C. Hertzman) and the Niagara Neighbourhood Association, both from Toronto; Friends of the Earth (prepared by T. Schrecker); DRI and ESSA; and the Canadian Environmental Law Association, which volunteered a notable essay. These have been consolidated in a single report called "Commentaries on Canadian Issues in Lead Contamination."

VII A bibliography of works identified by the Commission as relevant to its other reports, "Lead in the Environment: A Bibliography" (edited by Carol L. Murray).

Of these reports, I and II represent the considered views of the entire Commission. III is a personal appraisal by the Chief Scientist, but we have incorporated its conclusions into I. The rest of the reports have influenced our opinions.

In addition, the Commission submits the file of formal presentations made to it by its consultants, industry, other groups and a few individuals (Appendix II). These have been invaluable to our work. We do not always agree with the views expressed, but have considered them all carefully, and have drawn on many of them in our reports.

4. The Commission's Approach

The Commission's approach has been determined by the skills and experience of its members, and by a conviction that all of us share--that objective weighing of scientific evidence is a better way to judge an environmental issue than is an exchange of threats and rebuttals. We have sought a consensus on this issue. We found what we knew must be there: a common agreement that all Canadians must be protected against environmental toxins. But we also found widespread differences of opinion about how dangerous a toxin lead was in the special conditions of Canadian life. Our report addresses that question.

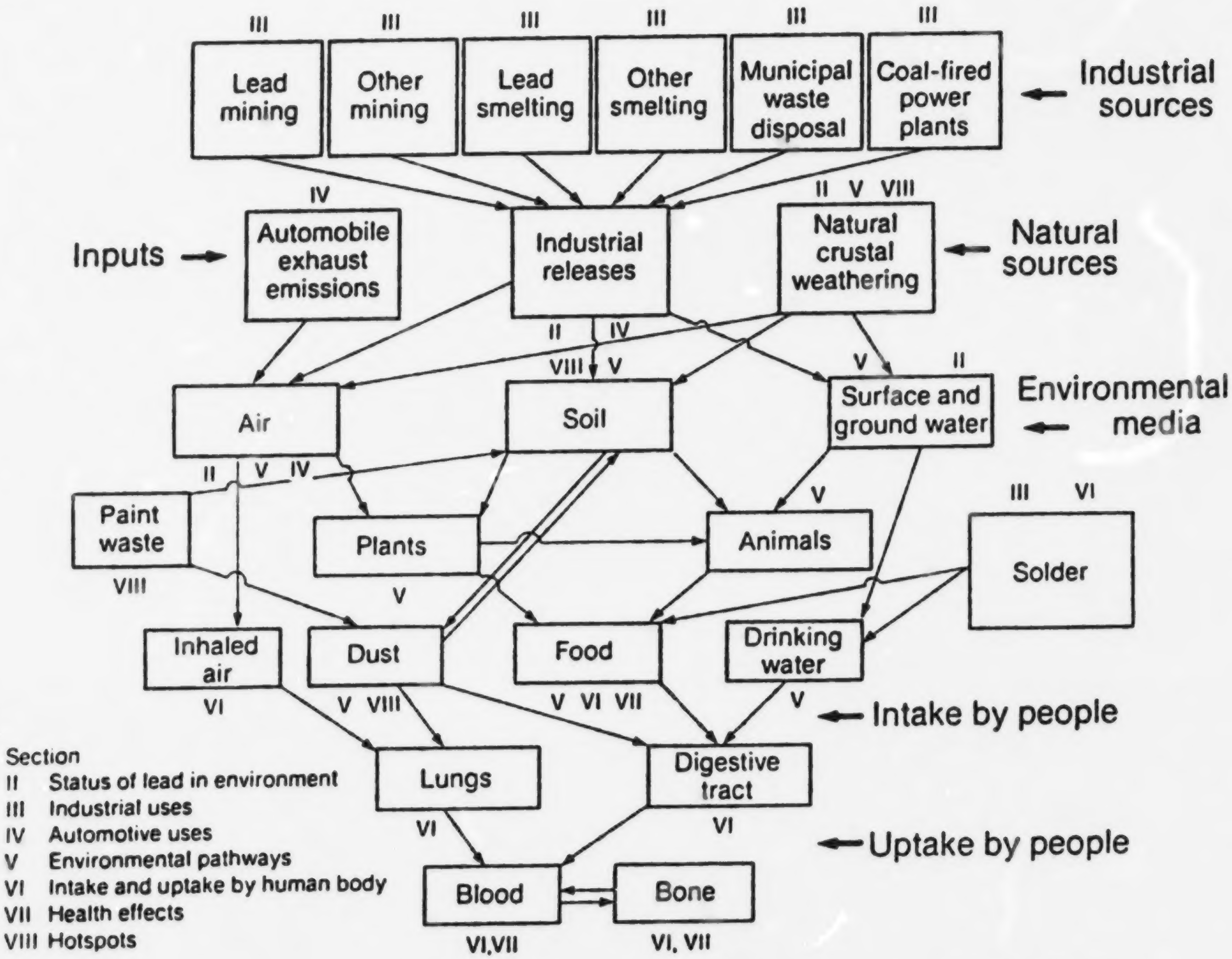
5. Structure of the Final Report

The report begins with a look at present-day concentrations of lead in the Canadian environment, together with an account of how these levels have changed over the past decade or so. The report ends with a look at lead's impact on human health, as revealed by recent surveys and research, and with a look at locations where lead has been an urgent issue.

In between, the text follows the scheme outlined in Figure I.1. Essentially this is an attempt to follow lead from its natural origins and the industrial sources that spread it through modern Canada until it reaches human targets via various intricate pathways. Within the human body it may create a variety of responses, some benign, others potentially dangerous. These are treated in detail.

The account then treats the hot-spot phenomenon in a series of studies of places in which contact between lead and human subjects has created perceived hazards and has led to demands for action. The text ends with a discussion of the Commission's findings, with a glance at the future. The set of appendices provides details referenced in the text, but not presented in full.

All conclusions and recommendations are included in the Chairman's Appraisal.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Industrial releases
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Bone
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Section
II Status of lead in environment
III Industrial uses
IV Automotive uses
V Environmental pathways
VI Intake and uptake by human body
VII Health effects
VIII Hotspots

Figure I.1. A summary diagram showing how lead moves from industrial and natural sources to the human target (with relevant sections shown).

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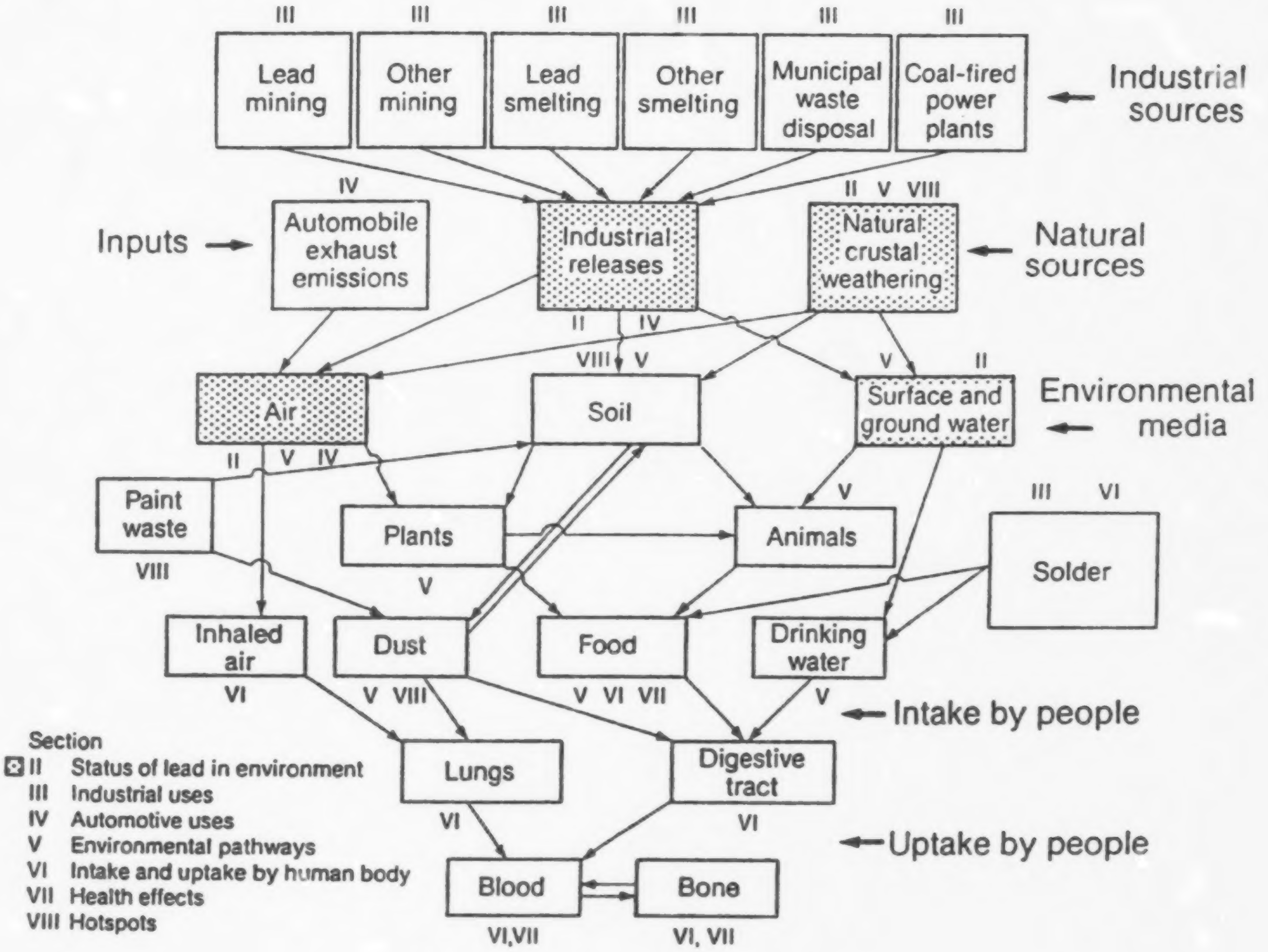
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Area covered in Section II

II Present Status of Lead in the Canadian Environment

How much lead is present in the accessible parts of the Canadian environment? To what concentrations of this toxic metal are we exposed?

Nriagu (1979) calculated that the global all-time lead production totalled some 260×10^6 t. He estimated that, since lead's earliest use, about 20×10^6 t may have passed into the world's active environment through the atmosphere, and that global natural emissions (volcanoes, dusts, forest fires, etc.) were outweighed 15-fold by anthropogenic sources.

About 40% of the total releases of lead to the atmosphere have occurred over the past 20 years, the largest single source being lead antiknock compounds used in motor gasolines. Nriagu (HWR) speculated that the exposure of contemporary Canadians to lead may be as much as 50 to 60 times greater than that of the precolonial population.

Except near smelters and industrial users, only a small amount of lead finds its way directly into soils, sediments and surface and ground waters. Most of the lead that reaches these sinks does so through atmospheric deposition. Several reports indicate that atmospheric lead concentrations have been decreasing over the past few years (Mahaffey et al., 1982; Southwood, 1983; EPA, 1986a).

It is much easier to monitor trends in concentrations of lead in air than in surface waters, sediments or soils. This has prompted attempts at regulation. In their efforts to control the absorption of lead by humans, some countries have set ambient air quality standards. In the US, EPA used three models to estimate the level of atmospheric lead at which 99.5% of children would have blood lead levels below the 25 µg/dL intervention established by the Centers for Disease Control (EPA, 1986b). The models indicated that an air lead level at least as low as the present 1.5 $\mu g/m^3$ standard, and perhaps no higher than 0.75 $\mu g/m^3$, would be necessary. To protect the same proportion of children at a blood lead level of 20 µg/dL, an air lead level no greater than 0.5-0.75 $\mu g/m^3$ would be required. A blood lead level of 15 µg/dL translates into modelled air lead levels of 0.25-0.5 $\mu g/m^3$. By contrast with the US standard, the CEC (1982) has set an ambient standard of 2.0 $\mu g/m^3$. Canada has not set any national standard, and has no current need to do so; Canadian air lead levels appear to be generally lower than those in other industrial countries. Ontario has set a 30-d geometric mean criterion for lead in air of 2 $\mu g/m^3$ and a 24-h criterion of 5 $\mu g/m^3$ (Heidorn and Rohac, 1984).

The concentration of lead in air depends very much, however, on site. Geography and climate influence values, as does the presence or absence of point sources of lead (which can give rise to hot spots, see Section VIII). The behaviour of particulate lead is also strongly dependent on particle size; particles less than 1 µm in diameter can be carried hundreds of kilometres, whereas larger ones settle close to the source. Thus, the larger particles that form in automobile exhausts will be deposited within 50 m of the roadway, whereas the smaller ones will mingle with particles of similar size from neighbouring streets, effectively blanketing large tracts of urban land. Davidson (PWR) examined the size distributions of airborne particulate lead. Most of the lead was found to be associated with the smaller particles (of submicrometre size) which are slow to fall onto natural surfaces, and may remain suspended for long periods of time.

Both Nriagu (PWR) and Davidson (PWR) stressed the importance of deposition of atmospheric lead, rather than its concentration in air. The rapid turnover rate of lead in the atmosphere (every 10 d, or about 40 times per year) implies that short-term or even 30-d means may not reflect the longer-term experience of the receiving surface. Air lead concentration is, therefore, not a good measure of the lead pollution burden in the environment. Nriagu (PWR) suggested that the fallout or deposition rate provides a more realistic estimate. Table II.I shows the deposition flux for some typical urban, rural and remote environments. Atmospheric lead has also been shown to be the principal source of lead in the open ocean, as well as an important source in coastal waters.

Truly representative measurement of air lead concentrations is further complicated by the siting of the monitoring recorders in the national survey networks. Many of the stations have been designed for specific monitoring requirements, such as an urban secondary lead smelter adjacent to a residential area. Hence their locations are not really the best for an ambient monitor. Monitors are often located for convenience as much as 10 m above a street. At this level the sensors will record concentrations far lower than kerb-side instruments. They may also be at differing distances from a street, again skewing the results (EPA, 1986a). The geometric mean of the observations is more satisfactory than the arithmetic mean, but the overall result may still be unrepresentative of general conditions.

More representative, however, is the trend revealed by the geometric means over time. As long as the network remains essentially unchanged (or only stations with a continuous record of data are used), the time trend information should be reliable.

Table II.I

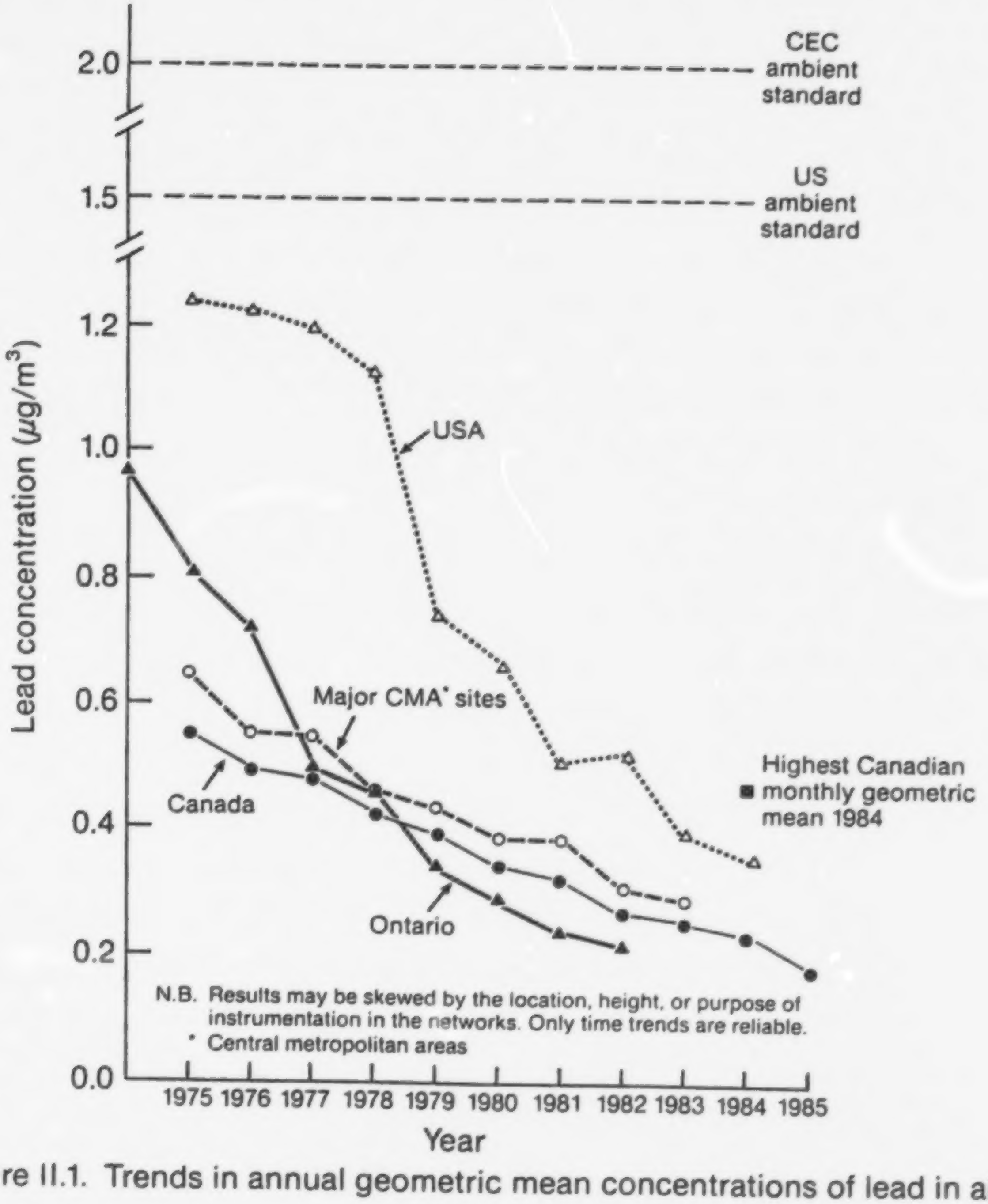
Atmospheric Fallout of Lead at Remote, Rural, Urban and Contaminated Areas

Site	Lead deposition flux ($mg/m^2/a$)
Near sources	
Toronto	> 6000
Toronto, near expressway	600
Urban	
New York City	547
Central Los Angeles	365-8000
Los Angeles suburbs	17-120
London, England	54
Madison, Wisconsin	> 25
Rural	
Northwest England	< 19.6
Delaware River watersheds	6.2
Upper Great Lakes Basin	12.0
Remote	
Scandinavian glaciers	4.2
Sierra Nevada Mountains, California	4.0
North-central Pacific Ocean	0.6
North Atlantic Ocean	1.4
Northern Greenland	0.04
Antarctica	0.01

Source: NAS, 1980

The ambient air lead concentrations since 1975 for Canada as a whole are compared with those for Ontario and Central Metropolitan Areas (CMA) in Figure II.1. The CEC and US ambient air quality standards are indicated, as is the highest monthly geometric mean for a Canadian National Air Pollution Surveillance (NAPS) Network station recorded in 1984 (0.65 $\mu g/m^3$). The record shows a clear decline in air lead levels throughout the period. Reductions in air lead concentrations have also been reported in the United States (EPA, 1986a) and the United Kingdom (Southwood, 1983). Though the reduction of lead levels in gasoline must have made a major contribution to reduction in air lead concentrations, it is quite clear from the comparison of the three countries shown in Table II.II that this was not the only factor responsible for the decline.

US ambient air lead data have been compared with the tonnage of lead consumed in gasoline between 1975 and 1983, and the two data sets are shown to have almost identical form (Figure II.2). In addition, Annest (1983) and Schwartz (HWR), using NHANES II (2nd National Health and Nutrition Examination Survey) data, reported strong correlations between blood lead concentrations and lead consumed in gasoline. Blood lead levels have declined quite generally, however, even in countries such as Britain and New Zealand which have had very little, if any, change in gasoline lead concentration or consumption (Table II.III). Indeed, Elwood (1983) and Elwood et al. (1985) found a 22% decrease in blood lead levels in an area of Wales over a period in which there were no significant overall changes in gasoline sales, air lead levels or lead concentrations in water. They suggested that, against such a background of falling blood lead levels, it would probably not be possible to attribute any



N.B. Results may be skewed by the location, height, or purpose of instrumentation in the networks. Only time trends are reliable.

* Central metropolitan areas

Figure II.1. Trends in annual geometric mean concentrations of lead in air.

Sources: EPA, 1984; Heidorn and Rohac, 1984; EPS, 1985 a, b.

Table II.II

Atmospheric Lead Reduction

Location	Time period*	% reduction in lead usage	% reduction in atmospheric lead
Canada	1970-1982	54	74
US	1975-1980	53	52
UK	1972-1982	9	50

* Time periods vary to match source data.

Sources: Southwood, 1983; EPA, 1986a; EPS, 1985a; PACE, 1985

Table II.III

Lead Used in Gasoline: Britain, Canada, United States

Year	Amount (x 10^3 t)		
	UK	Canada	US
1975	10.0	15.9	166
1982	9.2	9.1	60
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% change	8	43	64

Sources: Southwood, 1983; EPA, 1986a; PACE, 1985

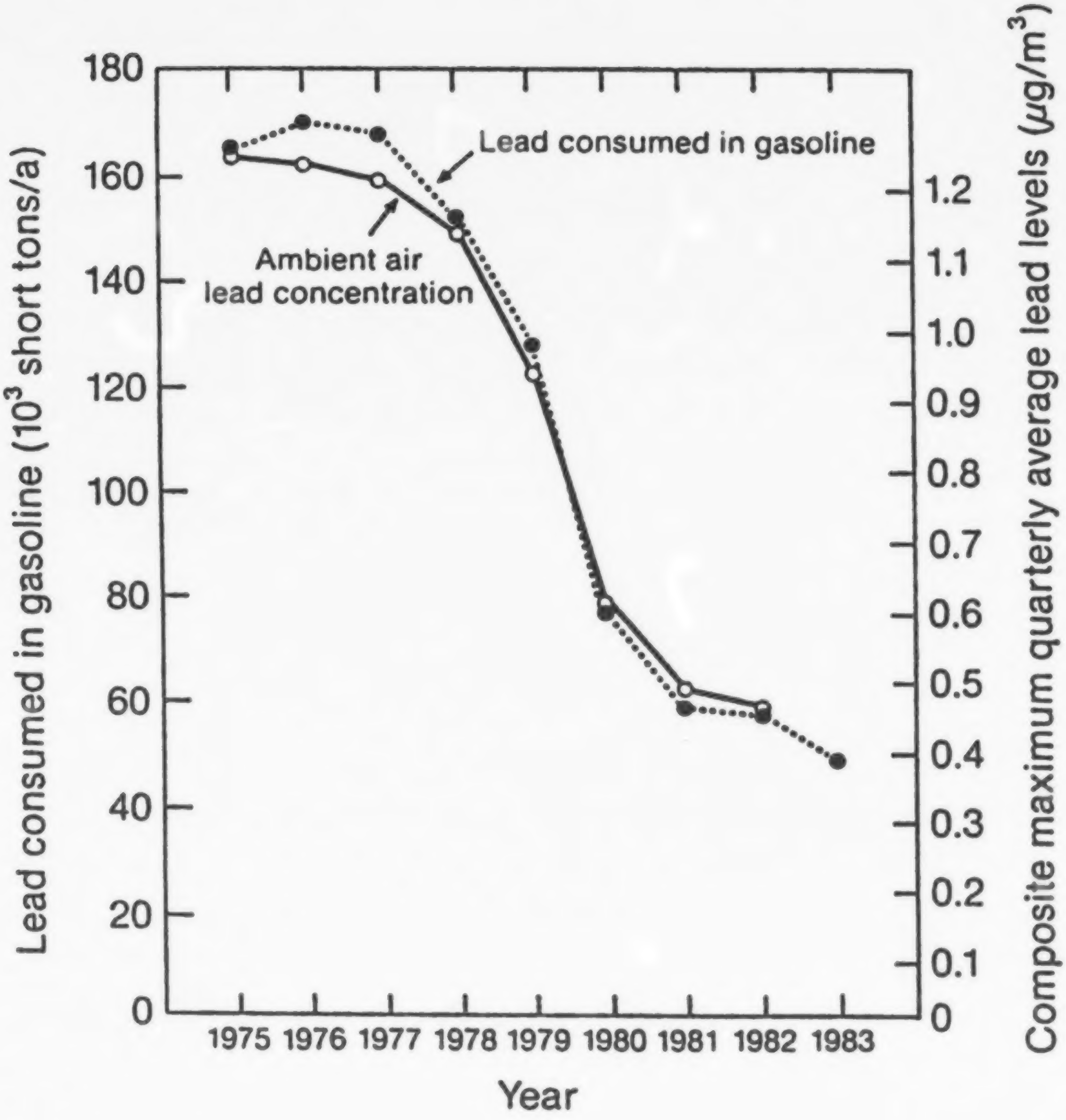

Figure II.2. Lead consumed in gasoline and ambient lead concentrations, US 1975-1983.

Source: EPA, 1986.

further effect on blood lead levels to changes in gasoline lead or air lead concentrations resulting from the 1986 UK gasoline reduction.

In contrast with air lead levels, there are few data on lead concentrations in water or in soil that can be used to establish overall national time trends. Indeed, because of the importance of regional and temporal conditions (rainfall, acidity, flow rate) as well as the chemical form in which lead is present in surface waters and soils, it is questionable whether aggregating measurements of trends in total lead concentration would have any meaning at all. An average of 12 years of aggregated data from 18 out of 22 major Canadian drainage basins is shown in Figure II.3 (Bird and Rapport, 1986). These data seem to show a general decline in lead concentration, but this cannot be regarded as more than an indication. More significant are the historical trends that can be inferred from sediments (Norton, PWR).

Cautiously, then, we can say that atmospheric lead concentrations in the Canadian environment appear to be declining. There are hints, but no more, that a similar decline is in progress in some of our water bodies. Similar improvements seem to be taking place in many countries--including some that have yet to take any action to reduce lead levels in gasoline.

However, we can say nothing about lead in the remaining water bodies, or in the soils and sediments. Logic suggests that there must be an accumulation in these reservoirs, as lead leaves the fluid media. Acquiring convincing figures to support this logic will, however, be extremely difficult.

In the report, we try to identify the pathways whereby lead moves through the environmental system towards its human targets. We also try to estimate the relative contributions of atmosphere, water and soil to human

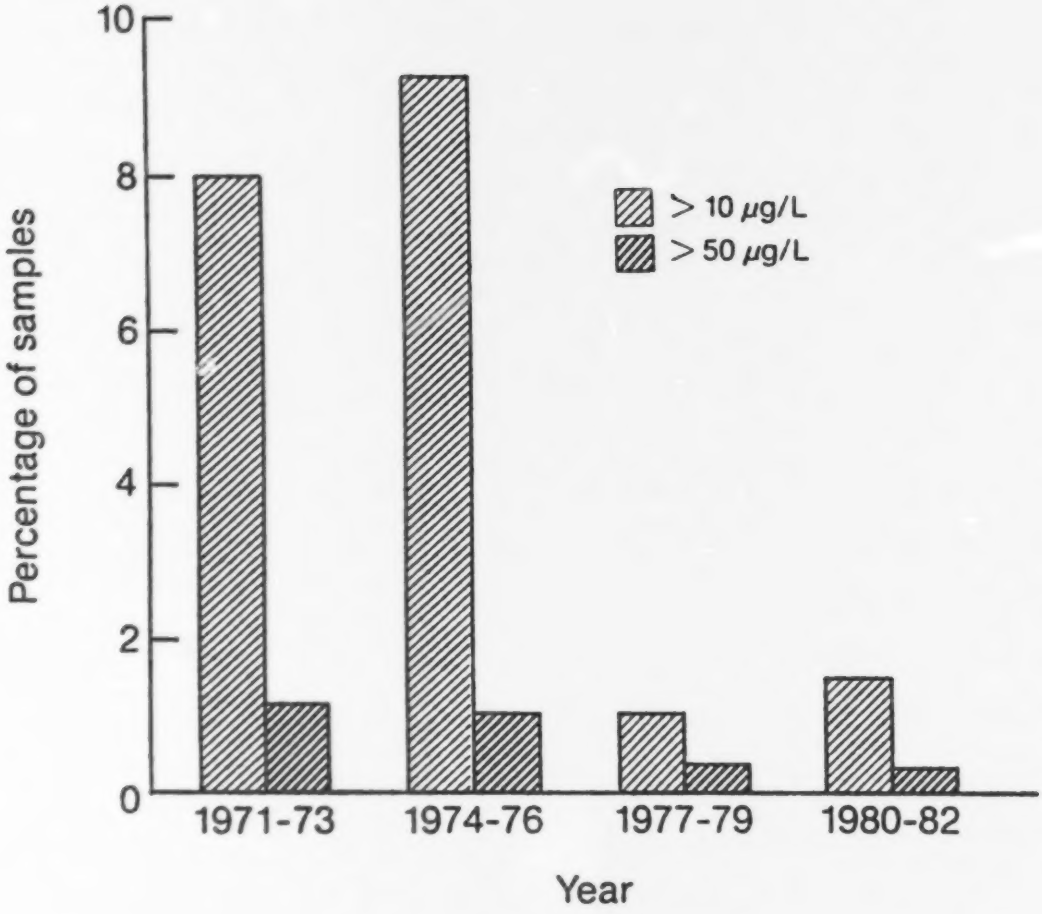

Figure II.3. Lead trends in Canadian natural waters 1971-1982.

Source: Bird and Rapport, 1986.

uptake of lead. None of these movements has been monitored to the extent reported for air. Instead, we shall be forced to rely on individual research results.

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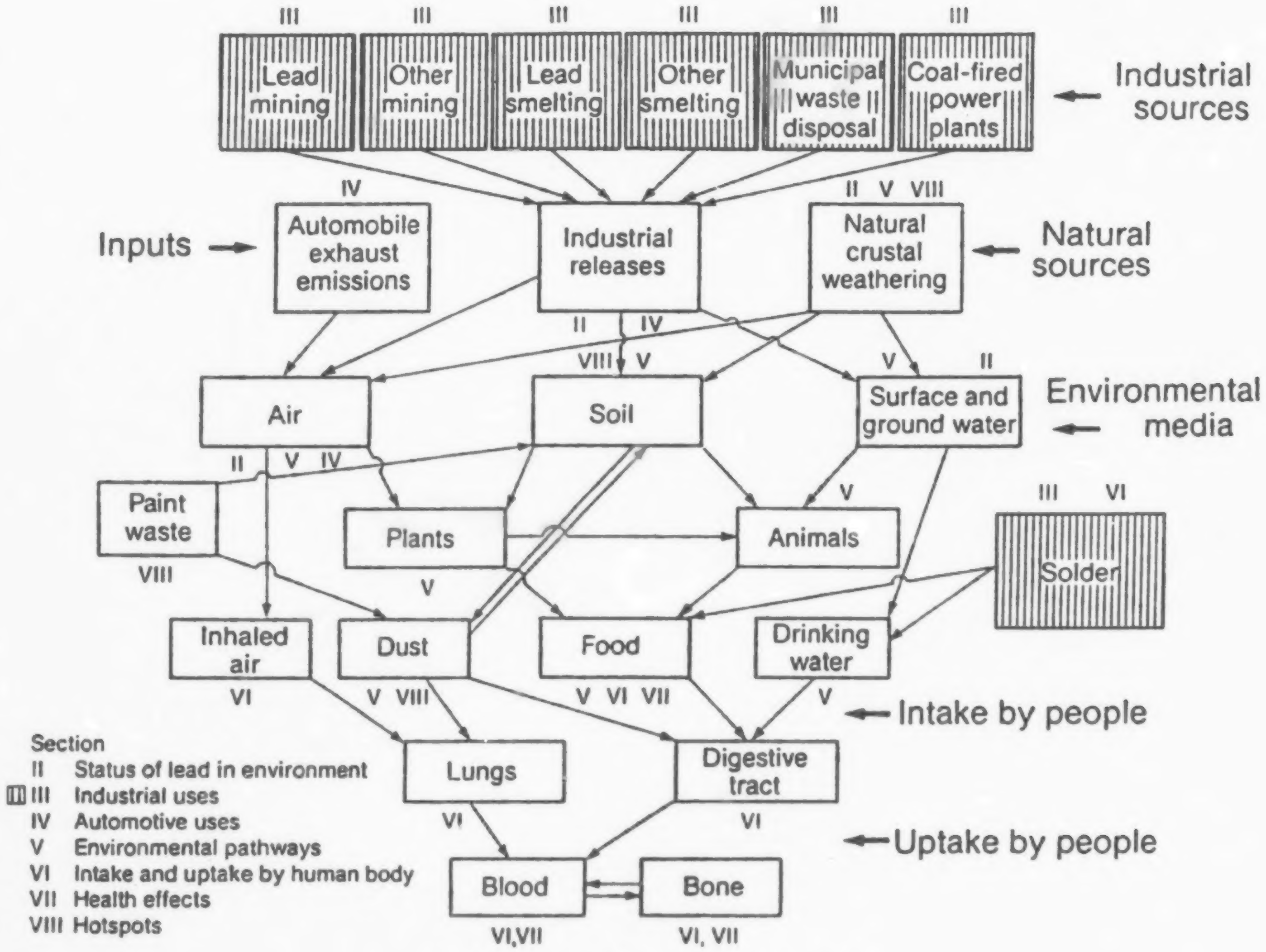

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V
IV
Paint waste
VIII
Plants
V
Animals
V
III
VI
Solder
Inhaled air
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Dust
V VIII
Food
V VI VII
Drinking water
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Intake by people
Lungs
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Bone
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II Status of lead in environment
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VIII Hotspots

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III Extraction and Industrial Uses of Lead

1. Introduction

Lead is the fifth most commonly used metal in the world, after iron, copper, zinc and aluminum. It has been used from earliest antiquity (it is mentioned in the Old Testament and was known to the Assyrians and the early Egyptians). It was used extensively by the Romans, mainly because of its malleability and its low melting point of 327°C. Other characteristic properties are its high density of 11.4 g/cm^3 (11.4 t/m^3) and its remarkable corrosion resistance towards most acids (except nitric acid), in particular sulphuric and hydrochloric acids. In nature it is found principally as lead sulphide (galena, PbS), and in Canada mostly in complex ores composed of lead and zinc sulphides, with small amounts of silver or silver sulphide minerals. Its corrosion resistance is partially due to its ability to form a large number of oxides, ranging from Pb_2O to PbO_2, which permit the formation of protective films on exposed surfaces (see Appendix VII, on the chemistry of lead).

Canada is the western world's third largest producer of mined lead, fifth largest producer of refined lead and second largest exporter of lead concentrates and refined metal. In 1984, Canadian primary lead production--lead obtained directly from ores--was 311 391 t, down from a 1971 peak of 400 000 t. Refined metal accounted for 174 987 t (Table III.I). Gross revenue of the primary mining and smelting industry was $280 million in 1984 (EMR, 1985). The industry directly provided employment for about 10 000 Canadians. In addition, the secondary smelting industry (which recycles scrap lead) recovered 79 000 t of refined lead, employing 700 people directly and 1400 others in spin-off jobs (Irwin, 1985).

Table III.I

Canadian Lead Production, Trade and Consumption

	Production (t)		Exports (t)			Imports (t)	Consumption* (t)
Year	All forms	Refined**	In ores and concentrates	Refined	Total	(Refined)	(Refined)
1970	353 063	185 637	186 219	138 637	324 856	1995	85 360
1980	251 627	162 463	147 008	126 539	273 547	1962	106 836
1984	264 301	174 987	114 208	124 149	238 357	6314	130 547

* Includes secondary lead.
** Primary lead only.

Source: Bigauskas, 1985

The fact that lead minerals occur in association with those of zinc, silver and copper (often called co-location) has helped to improve the international competitiveness of the mining and milling sector of the Canadian lead industry. Canadian smelting and refining competitiveness has been retarded by the failure of the primary industry to replace older, energy inefficient plants during the 1973-1979 period of peak profit margins. However, the modern flash smelting process only became available in the 1980's. Despite low prices and uncertain markets that discourage new investment, plans for plant renewal are proceeding at Trail with federal and provincial financial participation. Similar measures are undoubtedly required at Belledune, and presumably also call for government participation, given the financial climate. Furthermore, it is clear that fundamental changes in technology are needed to control overspills. Add-on pollution controls to outdated plants have been stretched to their limits and a more-of-the-same approach would be both ineffective and very costly.

The fall in profitability has been aggravated by structural changes in world mineral markets that are having major effects on the non-ferrous metals industry as a whole in this country, as well as by some factors that are specific to lead. These factors include energy conservation, reduction in the size of metal-intensive products such as automobiles, other technological developments, many of which have led to substitution of metals by other materials, and increasingly stringent and demanding environmental regulations. Consequently, the non-ferrous metals markets are expected to grow at about one-half of the annual rate of that experienced between 1945 and 1973 (EMR, 1985).

Furthermore, much of the expansion in world capacity since 1960 has been in developing countries, where the supportive policies of the governments of the major consuming nations have financed the construction of efficient, state-of-the-art plants at low interest rates in low wage-cost environments. The consequent oversupply and downward price trends for many commodities were also strongly affected by the recession of 1980-1982, particularly in the Canadian minerals industry, which exports about 80% of its products (Bigauskas, 1986a).

As the balance between supply and demand will take several years to re-establish--in most cases it is not expected to occur until the early 1990's--price recovery will be slow. Of particular concern to Canadian producers are a number of factors that will continue to distort the operation of an international free market. Many of their competitors are controlled or subsidized by government, and others are protected by tariff and non-tariff barriers, or are members of regional trading blocs. In addition, barter agreements involving suppliers with artificially low costs or protected by unrealistically low exchange rates are expected to become more frequent. Individually or in combination, all these factors will at least complicate, if not entirely frustrate, Canada's attempts to retain its traditional non-ferrous metals markets and to expand into new ones.

World lead consumption grew at an average annual rate of 3.7% between 1960 and 1973. Demand for most non-ferrous metals fell off abruptly with the international oil shortage that was provoked by the 1973 embargo. Lead, however, continued to maintain a 4.1% growth until 1979; this was sustained by increased demand for replacement batteries (EMR, 1985). Lead prices continued to rise to a peak of 73 cents (US) per pound ($1.61/kg) (constant 1983 dollars). This was helped by an oversupply of zinc, which, despite the highest-ever lead profit margins, restricted lead production because of the integrated nature of the production facilities for these two metals. The profit margins for the secondary smelters were even higher, and this sector increased its share from 33% of the western world's supply of refined lead to 45%.

At this point, reaction set in. Improved battery designs containing smaller proportions of lead led to a fundamental (or structural) decline in demand for the metal that will not be reversed by changes in purely economic conditions. Another structural change came about from increasingly widespread restrictions on the use of tetraethyl lead (TEL) and tetramethyl lead (TML) antiknock agents in gasoline. These were in force by 1980 in the United States, Canada, the European Communities, Japan, Sweden and Switzerland. They were accompanied by a trend towards smaller, more fuel-efficient vehicles, which reduced overall gasoline consumption.

The most recent blow to the industry was the 1980-1982 recession and the slow recovery of demand afterwards. Because lead and zinc ores have to be mined and processed together, the demand for zinc stimulated by the economic recovery led to the already large lead inventories increasing until 1983, and prices fell to 22 cents (US) per pound (48.5 cents per kilogram) (constant 1983 dollars). The average price level in 1984 was 25.5 cents per pound, but this fell back to 19 cents in 1985. The mid-1986 price level was around 22 cents (US) per pound (48.5 cents per kilogram).

Unlike the mining and milling sector, Canadian smelters and refiners have one of the world's highest cost structures (Figure III.1). To maintain their market shares, the smelters and refineries will have to become more

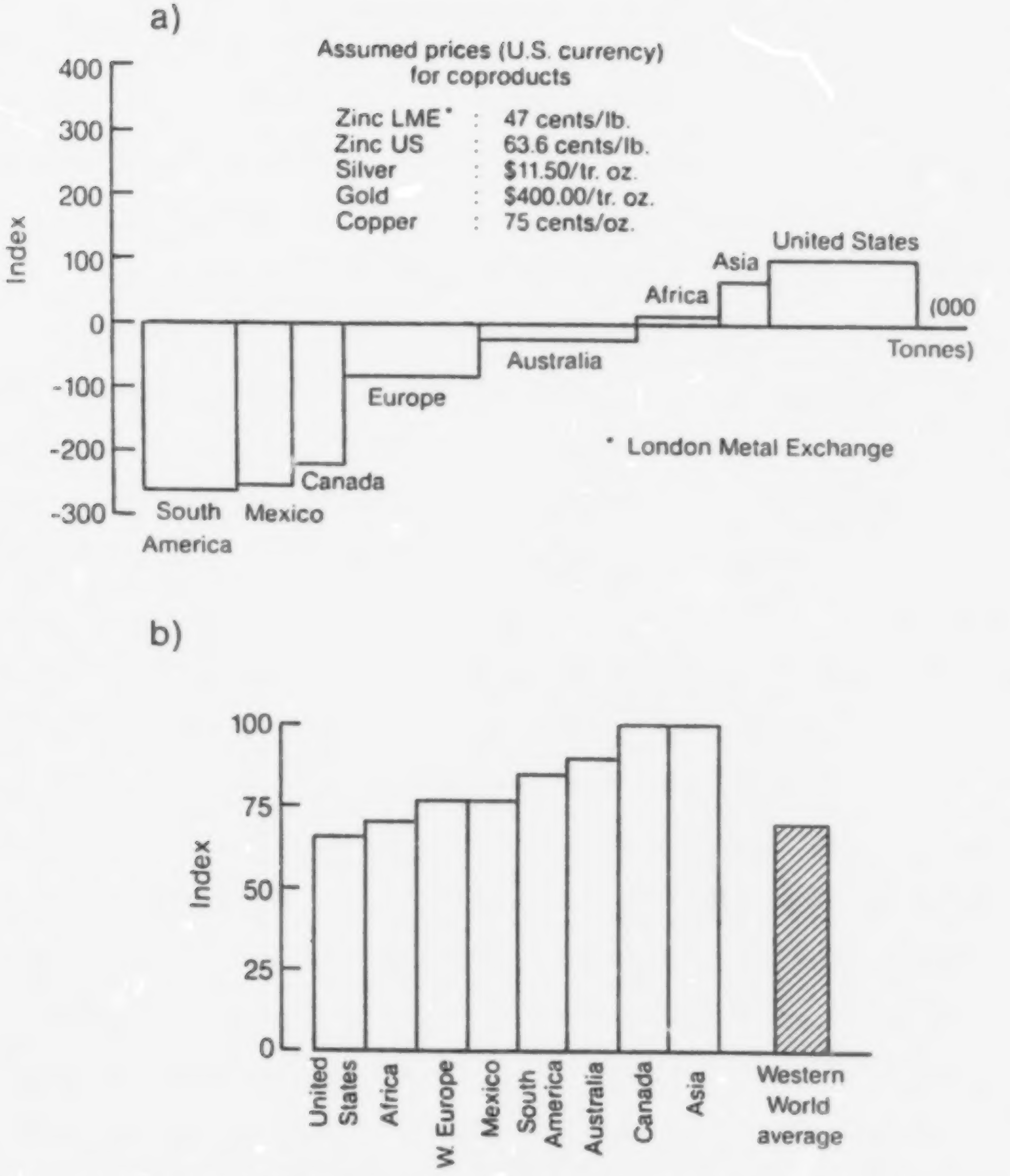


Figure III.1. Competitive cost rankings of (a) major lead mine producers, and (b) major lead smelting and refining regions, 1984.

Source: EMR, 1985.

competitive--and not marginally so, as they are now. This will entail capital investments in the order of several hundred million dollars (Bigauskas, 1986a). It does not seem likely, under the present circumstances, that the integrated producers or their parent companies could easily raise or accommodate such expenditures, given their capital and debt structures. However, Bigauskas (1986a) believes that such a strategy is essential to ensure the survival of the primary lead industries, which are forced to operate under tariff and non-tariff barriers in a competitive and declining market. The Canadian and British Columbia governments have recently agreed to invest in the lead smelter modernization at Trail (the smelter dates from 1906) by the purchase of $79 million and $55 million, respectively, of redeemable preferred shares to be issued by Cominco Ltd. The decades-old, high-cost smelter will be replaced by a low-cost, state-of-the-art smelter that will help the plant's competitiveness in world markets. In addition to substantial production costs, it should also mean improved environmental and workplace protection.

The main export markets for Canadian lead have been the United States, Europe and Japan. All are expected to be highly competitive for the foreseeable future. The US market is expected to grow slowly, but pressures already exist to restrict its accessibility to all imports. European projections indicate a stagnant lead market, perhaps starting to grow slowly, and it will remain difficult to make inroads into a Japanese market that is unlikely to be relieved of its traditional protectionist policies.

Canadian lead consumption is much lower than its national production. In 1984, 130 547 t of refined primary and secondary lead were used in this country, and the remainder, almost half the total production, was exported (Bigauskas, 1985). Most of the domestically produced lead is used by the battery industry; other large consumers are the manufacturers of paints and coatings, tetraalkyl lead gasoline additives, alloys and solders (Figure III.2).

There was a marked annual increase in the consumption of refined lead in Canada between 1970 and 1984 (45 000 t). During this period the production of primary refined lead decreased by 10 650 t, and exports by 14 488 t. After taking account of a small increase in imports, the data in Table III.I show that the increase in consumption could only have been met by much more effective recycling of secondary lead.

2. Waste Discharges

In 1982 an estimated 73 000 t of lead were discharged into the Canadian environment, mostly as solid waste (61 000 t) (Jaques, 1985). By far the largest proportion is metallurgical waste. Lead contained in slags is estimated to account for about 17 000 t; these slags are very unreactive, and the rates at which they leach lead to the soil are believed to be very low. Another 38 000 t of lead, mainly unchanged (and unreactive) lead sulphide, are discharged as concentrator tailings from the initial ore flotation.

Of 562 t of liquid effluents, 412 t of lead were contained in municipal sewage (73%) and 87 t (15%) came from mining, milling, smelting and refining of metals.

More significant are the emissions of lead particulates to the atmosphere (11 446 t in 1982). The largest single source was gasoline lead--particulate products of small particle size (less than 1 µm diameter) resulting from the combustion of TEL and TML antiknock agents (see Section IV). This accounted for 7278 t--63% of the 1982 emissions--but gasoline lead emis-

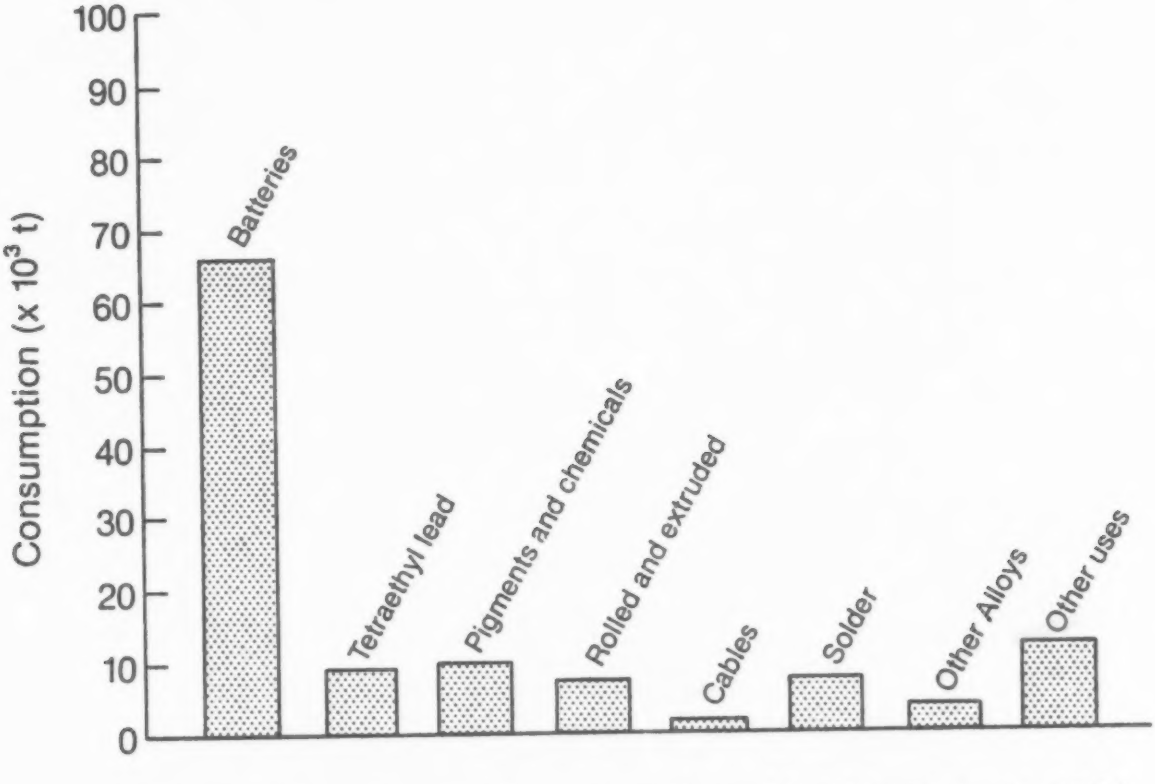


Figure III.2. End-use consumption of lead in Canada, 1984.

Source: Irwin, 1985.

sions are falling annually under regulatory pressure (see Figure IV.1, Section IV). Mining and refining of non-ferrous metal ores that contain lead as an impurity--particularly copper, nickel and zinc ores--were responsible for 18% of the lead emitted to the atmosphere in 1982, a greater proportion than for the lead production industry itself (9%). Emissions from iron and steel production facilities were also large (5%). Though the concentrations of lead present in the ferrous and non-ferrous ores are small, the extremely high ore tonnages milled yield large quantities of lead. Particle sizes related to this type of emission are usually in the 10- to 500-μm range.

A good example is the on-site disposal of baghouse dusts from electric furnace operations in the steel industry. These are extremely fine, and contain large quantities of zinc (about 20%) and lead (about 10-15%) from the recycling of steel scrap. It is estimated that more than 200 000 t of these dusts are stored in Ontario and Quebec (Gauvin, 1986).

Appreciable quantities of lead and other toxic materials such as polychlorinated biphenyls (PCB's) occur in waste lubricating oils. About 18% of the waste oil is collected in Canada, and half of this is re-refined (Fisher, 1985). The remainder is either incinerated, annually emitting about 130 t of lead to the atmosphere, or spread on roads to control dust. Lead is easily mobilized from this road oil, and can wash into the soil or nearby water bodies (Metzler and Jarvis, 1985).

Lead filler has been used for automobile body repairs for many years, though it is now being phased out. This material is painted over, and is ignored when automotive scrap is recycled, passing up the stack and making a significant contribution to the lead emissions from ferrous foundries. A further contribution comes from any batteries not removed before the car is crushed. The secondary lead industry is responsible for only 0.12% of the atmospheric emissions--some 14 t (Jaques, 1985). This amount is offset by 1500 t of solid waste (Irwin, 1985), in large measure lead in slags, though there are also contaminated casings and other used materials. Baghouse dusts and fly ash are recycled, and the quantities collected are not included in the above figures (Jaques, 1985).

The methods developed for waste handling and control by the primary and secondary lead industries have been adapted by industrial users of lead. Dusts and fumes are collected in baghouses or precipitators by efficient ventilation systems, and workers are required to wear masks and other protective clothing in high-exposure areas.

3. The Primary Lead Industry

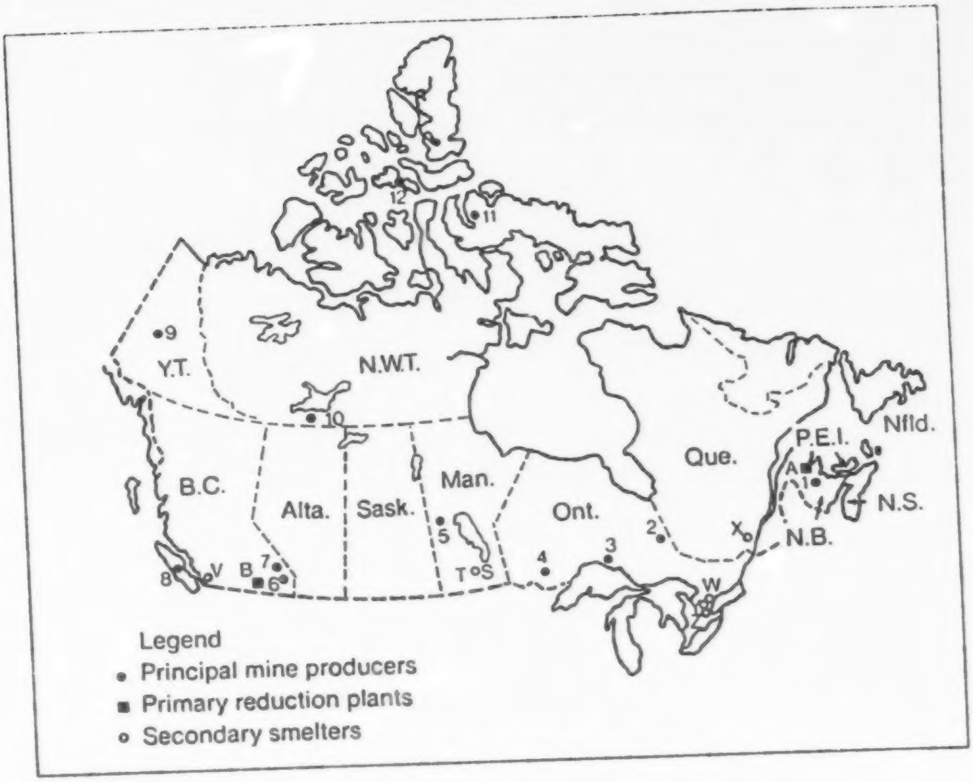
Ores exploited in Canada range from 2 to 5% lead by mass. They are generally sulphides of lead and zinc, usually with some silver, copper or iron (EMR, 1985). Proven and indicated lead ore reserves in Canada amount to 17 x 10^6 t of contained metal, which is about 13% of the lead reserves of the non-socialist countries. Most of the lead produced in Canada is a co- or by-product of zinc extraction. Canada's 56 x 10^6 t of zinc reserves rank first in the western world, and Canada also has the highest zinc mine production. The close association of lead and zinc means that zinc production requires by-product lead to be mined and at least processed to a concentrate, so that lead production cannot simply be curtailed or expanded in response to the demand for the metal.

Canada's producing lead concentrators are located in New Brunswick, Ontario, Manitoba, British Columbia, Yukon Territory and the Northwest

Territories. Though several mines have closed, either temporarily or permanently, the Cyprus Anvil Mine in the Yukon reopened in mid-1986. Only two large plants produce refined lead. These are located at Belledune, NB (Brunswick Mining and Smelting Corporation, controlled by Noranda Inc.), and at Trail, BC (Cominco Ltd.) (Figure III.3). The Trail smelter is older, but has been modified several times since its construction in 1906. The smelter at Belledune was built between 1964 and 1966 to treat the complex local ores. Since being modified in 1971, the smelter no longer handles lead/zinc ores, but only smelts lead chiefly from concentrates shipped in from mines in the region. The nature of the concentrate used in this operation is the primary cause of its high operating cost.

The treatment and refining processes are illustrated in Figure III.4. The ore is first crushed and ground, and the various minerals present are separated from the rock-forming minerals (gangue) by differential flotation to produce a lead or lead-silver concentrate. The concentrates are either exported or shipped to one of the Canadian primary smelters, where they are first sintered to remove sulphur (as sulphur dioxide) and then converted to molten lead bullion by roasting with coke and fluxing agents to form a slag that collects some of the dissolved impurities. The molten bullion is then further refined to remove copper, bismuth, antimony and arsenic, and also to recover doré metal, which is 99.6% silver. The sulphur dioxide generated is converted to sulphuric acid, which is used in nearby plants to make ammonium phosphate fertilizers.

Lead-containing dusts constitute hazards in the mining, milling and transportation operations, as does 'fume' in the smelting process--finely divided oxide particles of diameter 1 μm or less, which are easily inhaled.


Y.T.
N.W.T.
B.C.
Alta.
Sask.
Man.
Ont.
Que.
P.E.I.
Nfld.
N.S.
N.B.
1
2
3
4
5
6
7
8
9
10
11
12
A
B
S
T
V
W
X
Legend
• Principal mine producers
■ Primary reduction plants
○ Secondary smelters

Principal mine producers
(numbers refer to locations on map above)

1. Brunswick Mining and Smelting Corp. Ltd.
2. Kidd Creek Mines Ltd.
3. Noranda Inc. (Geco Division)
4. Mattabi Mines Ltd.
   Noranda Inc. (Lyon Lake, "F" Group)
5. Hudson Bay Mining and Smelting Co. Ltd.
6. Cominco Ltd. (Sullivan mine)
7. Dickenson Mines Ltd. (Silmonac mine)
8. Westmin Resources Ltd. (Lynx and Myra, H.W.)
9. United Keno Hill Mines Ltd. (Elsa)
10. Pine Point Mines Ltd.
11. Nanisivik Mines Ltd.
12. Cominco Ltd. (Polaris mine)

Metallurgical plants

A. Brunswick Mining and Smelting Corporation Ltd.
   Smelting Division, Belledune
B. Cominco Ltd., Trail

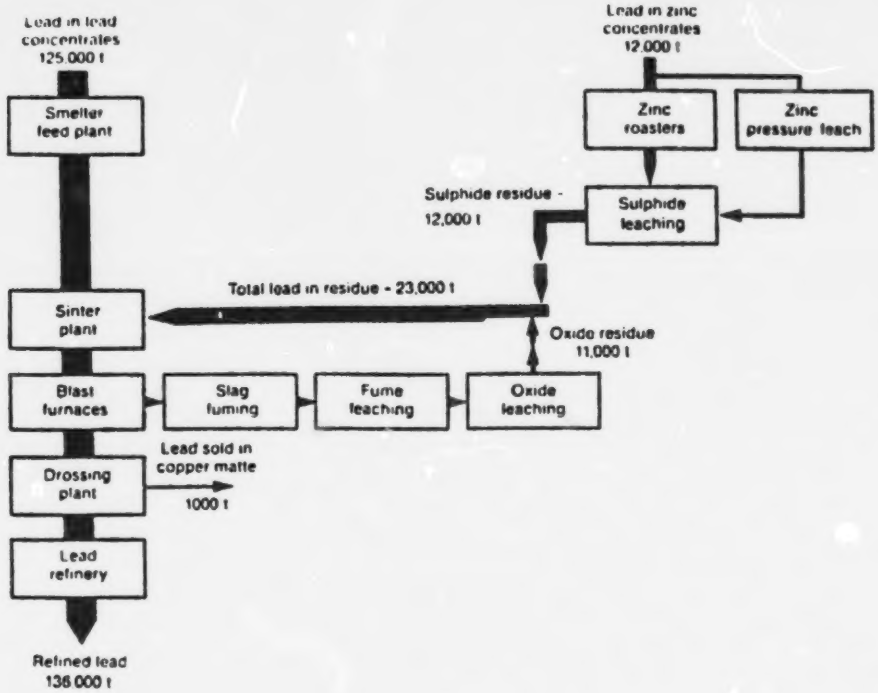
Secondary smelters

S Canada Metals Ltd., Winnipeg
T Northwest Smelting Ltd., Winnipeg
V Metalex Ltd., Vancouver
W Canada Metals Ltd., Toronto
X Nova Pb Inc., Montreal
Y Toronto Refiners and Smelters Ltd.
Z Tonelli Canada Ltd., Mississauga

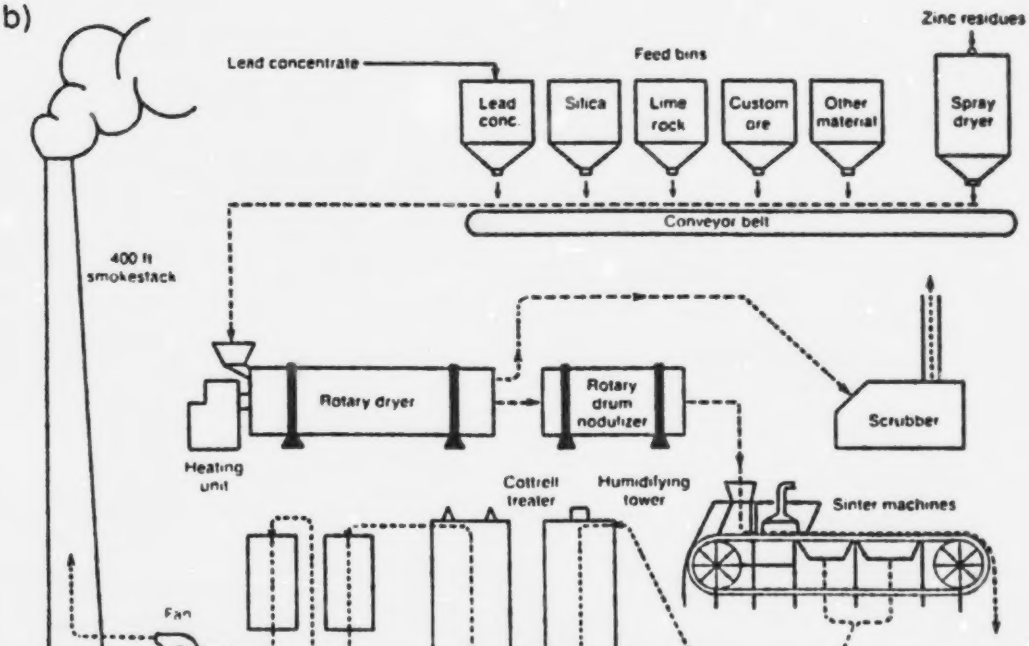
Figure III.3. Principal mine producers, metallurgical plants and secondary smelters in Canada, 1985.

Source: Bigauskas, 1985.

a)


Lead in lead concentrates 125,000 t
Smelter feed plant
Lead in zinc concentrates 12,000 t
Zinc roasters
Zinc pressure leach
Sulphide residue - 12,000 t
Sulphide leaching
Total lead in residue - 23,000 t
Sinter plant
Oxide residue 11,000 t
Blast furnaces
Slag fuming
Fume leaching
Oxide leaching
Lead sold in copper matte
1000 t
Drossing plant
Lead refinery
Refined lead 136,000 t

b)


Zinc residues
Feed bins
Lead concentrate
Lead conc.
Silica
Lime rock
Custom ore
Other material
Spray dryer
Conveyor belt
400 ft smokestack
Rotary dryer
Rotary drum nodulizer
Scrubber
Heating unit
Cottrell treater
Humidifying tower
Sinter machines
Fan
Sulphur dioxide absorption
Fan

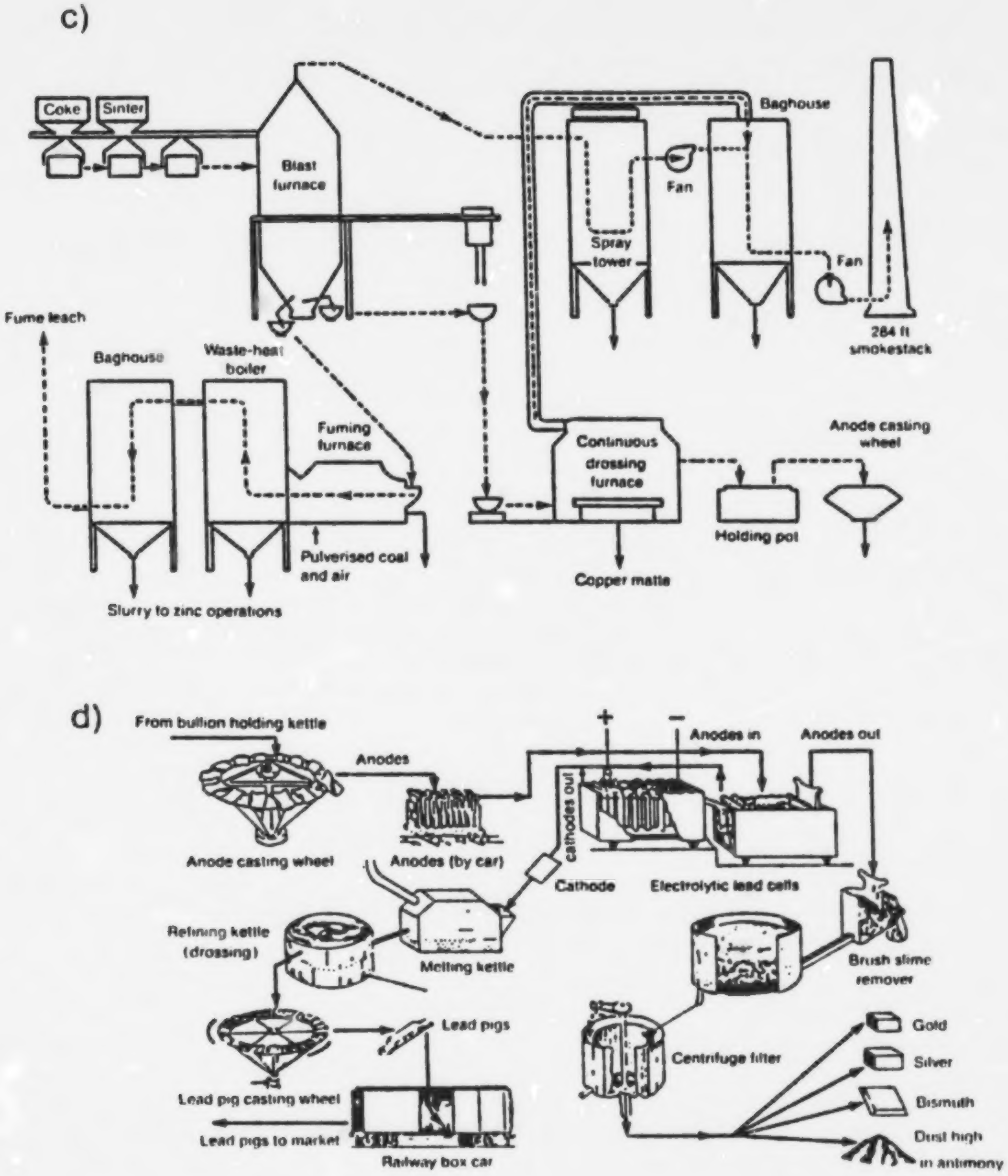

c)
Coke
Sinter
Blast furnace
Baghouse
Fan
Spray tower
Fan
284 ft smokestack
Fume leach
Baghouse
Waste-heat boiler
Fuming furnace
Pulverised coal and air
Slurry to zinc operations
Continuous drossing furnace
Copper matte
Holding pot
Anode casting wheel
d)
From bullion holding kettle
Anodes
+
−
Anodes in
Anodes out
cathodes out
Anode casting wheel
Anodes (by car)
Cathode
Electrolytic lead cells
Refining kettle (drossing)
Melting kettle
Brush slime remover
Lead pigs
Centrifuge filter
Gold
Silver
Bismuth
Dust high in antimony
Lead pig casting wheel
Lead pigs to market
Railway box car

Figure III.4. Primary reduction of lead. a) Trail lead operations b) lead sintering c) lead smelting d) lead refining.

Source: Cominco, 1985.

Both Canada's primary smelters are located in thinly populated areas, and most of the mines that feed them are in quite remote places. The surrounding regions are heavily dependent on the mines and smelters for employment. Brunswick Mining and Smelting Corporation Ltd. employs 601 people at the Belledune smelter and 100 more in the fertilizer plant; Cominco Ltd. has 2700 workers at Trail and 1000 at Kimberley. The smelter operations pose potential occupational hazards to the workers and their families in the nearby townships. The mining and smelting companies have relied on dust collection, ventilation and other safety programs such as baghouses and compulsory wearing of masks in high-exposure areas in the plants to protect workers' health, which is closely monitored. Blood lead determinations are done routinely, and workers are removed from high-exposure areas if their blood lead levels approach the regulated intervention level of 60 µg/dL in British Columbia, or the union-negotiated level of 70 µg/dL in New Brunswick. Provincial inspectors monitor the mining and smelter operations closely to enforce government safety and health regulations. Lead emissions and effluents from the primary smelting industry are included in Tables III.II and III.III.

The environmental problems arising from the Kimberley-Trail and Belledune-Bathurst plants are discussed in Section VIII.

4. The Secondary Lead Industry

The Canadian secondary lead industry recovers and recycles between 60 000 and 80 000 t of lead each year. The industry processes about 15 000 used lead-acid batteries per day, which provide between 80 and 90% of its raw material. Most of the remainder is made up of drosses (lead-bearing scum from the surfaces of melting pots). Cable sheathing, solders, pipes and other lead

Table III.II

Lead Content of Effluents to Receiving Waters, Canada, 1982

Sector	Effluent (t)	% of total
Industry		
Municipal sewage	412	73
Mining and milling	49)	15
Smelting and refining	29)	
Primary iron and steel	9)	
Chemical manufacture	37	7
Pulp and paper	9	2
Metal plating	5	< 1
Battery manufacture	4	< 1
Metal fabrication	2	< 1
Oil refineries	2	< 1
Foundries	1	< 1
Miscellaneous	4	< 1
Total	562	100
Province		
Quebec	250	44
Ontario	125	22
British Columbia	62	11
New Brunswick	46	8
Alberta	21	4
Nova Scotia	20	4
Territories and other provinces	38	7
Total (all provinces and territories)	562	100

Source: Jaques, 1985

Table III.III

Atmospheric Emissions of Lead, Canada, 1982

Source	Emissions (t)	% of total
Gasoline (including alkyl lead manufacture)	7 278	63
Primary production of metals		
Lead and zinc	1 268*	11
Other non-ferrous metals	1 706	15
Iron and steel	600*	5
Secondary lead smelters	14	0.1
Industrial uses		
Batteries	20	0.2
Other lead industries (including paints and pigments)	27	0.2
Metal foundries and fabricators	301	3
Cement manufacture	6	< 0.1
Metallurgical coke production	2	< 0.1
Energy production (stationary sources)	25	< 0.1
Waste incineration	199	2
TOTAL	11 466	99

* Emissions of lead from iron and steel plants are comparable with those from lead plants.

Source: Jaques, 1985

wastes are also handled. A typical plant flow diagram is shown in Figure III.5.

Scrap dealers collect batteries and other lead materials and deliver them to the plant. In a modern smelter, the used batteries are first processed through a separation unit, in which sophisticated battery breakers remove the lead materials from the casings and separators and neutralize the battery acid. The lead-containing mud is removed from the casing, and the acid is discharged to the sewers after dilution to concentrations that must conform with provincial or municipal regulations. The polypropylene and hard rubber casings, together with the separators, are washed and sold if reusable, or disposed of to an approved landfill site.

The lead-containing material is then smelted to produce an impure metallic lead. Most secondary smelters use rotary reverberatory furnaces or blast furnaces, which produce a furnace metal containing about 1.75% antimony and a slag. Entrapped lead in the slag is chemically inert, and is disposed of as landfill.

The impure metal is then refined and alloyed to customer specifications, the drosses produced in this process being recycled back through the smelting operation (Irwin, 1985).

The secondary lead industry is by nature an urban resident. It needs to be close to a very large source of its raw materials, and at a transportation hub that allows batteries and other waste lead to be economically collected from as far as 1500 km away. Its processes, however, have been responsible for contamination of the urban environment, particularly in Toronto, where two secondary smelters are located in close proximity to residential areas (see Section VIII). In recent years, as understanding of the health effects of lead

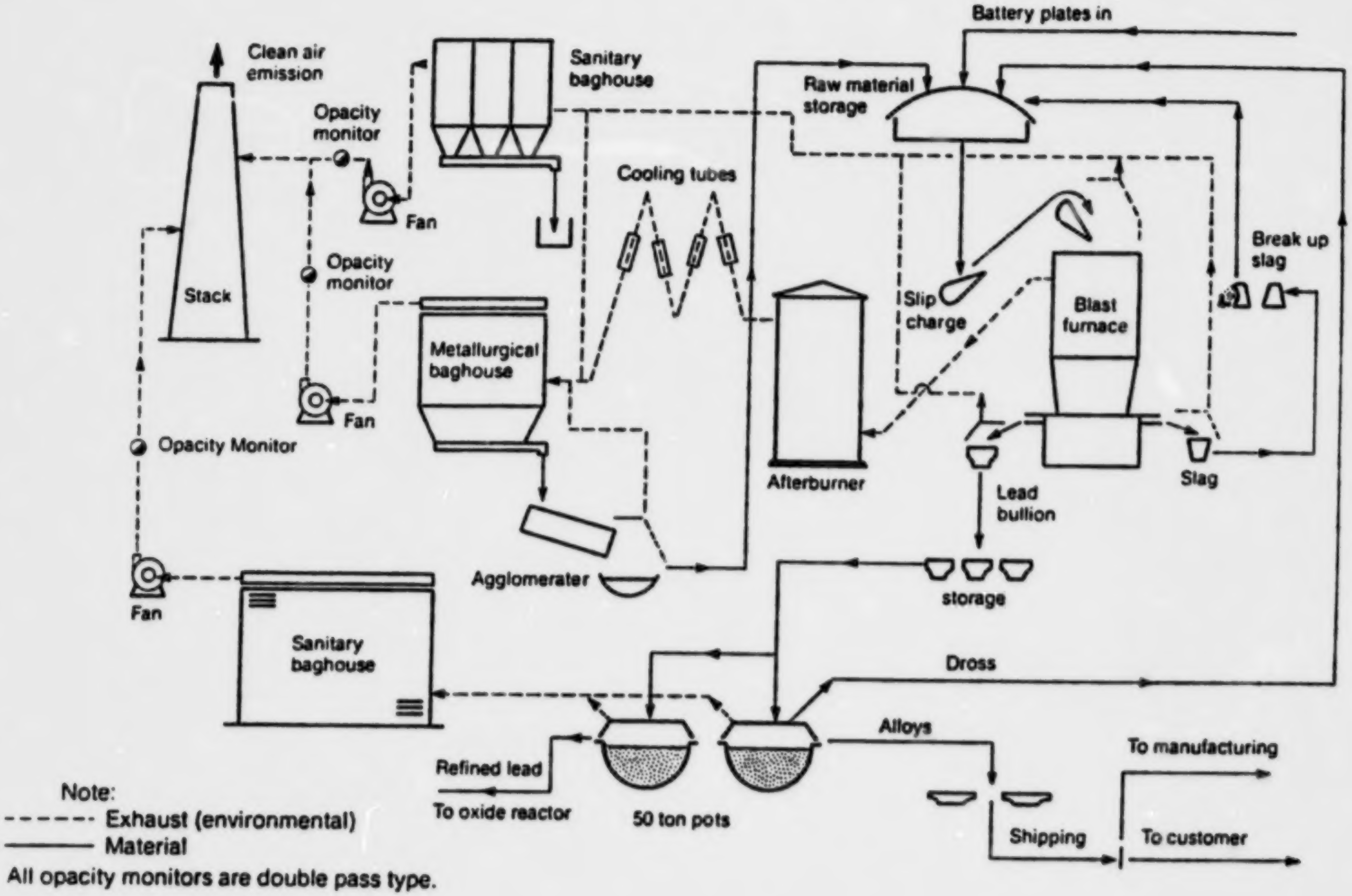

Battery plates in
Clean air emission
Sanitary baghouse
Raw material storage
Opacity monitor
Fan
Cooling tubes
Break up slag
Opacity monitor
Slip charge
Blast furnace
Stack
Metallurgical baghouse
Fan
Opacity Monitor
Afterburner
Lead bullion
Slag
Agglomerater
storage
Fan
Sanitary baghouse
Dross
Alloys
To manufacturing
Refined lead
To oxide reactor
50 ton pots
Shipping
To customer
Note:
----- Exhaust (environmental)
——— Material
All opacity monitors are double pass type.

Figure III.5. Secondary lead smelting.

Source: Irwin, 1985.

has grown, the industry has installed more efficient baghouses to control its emissions, as well as enclosed hoods on melting pots and better ventilating systems, both to protect the workers and to prevent the escape of lead-containing particulate matter from the plant. Some plants now have negative internal pressure systems to prevent dust from escaping through doors. Yard work has also been organized to avoid disturbance of dry materials, and stocks are now kept in properly vented indoor stores. Lead emissions from the secondary smelters in 1984 were estimated to be 15.8 t, based on production of 79 000 t (Irwin, 1985) and an emission factor for the industry of 0.2 kg/t (Jaques, 1985).

The impressive results achieved by the industry's improved awareness of health and environmental concerns, and the emissions controls that have been introduced, have entailed large capital and operational expenditures. The secondary refiners tend to be mature businesses with older plants--there is no Canadian secondary smelter that has been designed and built throughout as a modern, state-of-the-art operation. In the current period of depressed metal prices, the costs of new investment are so great that insistence on relocation away from residential areas, which would entail clean-up, site decontamination and plant replacement, would lead to virtual shut-down of the industry. Despite the fact that oxygen-enriched furnaces and low-temperature recovery processes are more efficient as well as environmentally benign, their installation would not confer a financial benefit on existing operations in the present economic situation.

The secondary lead industry is one of the most efficient of all recycling industries; it claims to have achieved a 98% recovery efficiency in the recycling of its raw materials. Lead wastes are removed from garages, streets, factories and storage areas at no cost to local authorities, and are reprocessed into a useful commodity; the alternative would be indiscriminately dumped batteries with toxic and dangerous contents (particularly sulphuric acid) that would have to be collected at public expense. Accumulation and dangerous dumping of batteries in municipal garbage are already becoming problems in parts of the United States, where the currently depressed price of lead has forced over half the secondary smelters operating in 1980 to close. The low price of lead (48.5 cents (US) per kilogram, down from a 1979 high of $1.61/kg) also does not provide any real incentive to collect batteries for recycling. In 1980, approximately 94% of spent lead-acid batteries in the United States were delivered to secondary smelters for reclamation, compared with an estimated 66% in 1985 (Palmer and Sappington, 1986). Some of the rest are actually exported to Canada.

Demand for lead from the US battery market amounted to 714 000 t in 1985, about 70% of the total demand; this represented a decline from 763 000 t in 1984, and inventories increased substantially. This drop was reflected in the secondary smelting industry, which saw total US output fall well below the 1984 level of 618 000 t, as 308 000 t/a of capacity was permanently or indefinitely shut down. US recycling capacity has fallen from 1.1 x 10^6 t in 1981 to 600 000 t in 1986, only two-thirds of which is operational. Improvement in the future is unlikely, as secondary smelting and battery breaking were effectively classified as hazardous waste under the Resource Conservation and Recovery Act, requiring those companies to have expensive environmental impairment liability insurance, as well as special treatment and storage facilities. Should this trend of plant closures continue, Palmer and Sappington (1986) estimate that by 1990 nearly 1.4 x 10^6 t of spent lead-acid batteries will have been improperly disposed of during the decade. Indeed, the newest smelter in the United States announced its intention to close in 1986, because of its inability to obtain insurance.

Three smaller smelters in Canada have closed since 1984, also as a result of price fluctuations. Currently, three are operating in the Toronto area, two in Montreal, two in Winnipeg and one in Vancouver. Of these, three are located close to residential areas. If the secondary smelting of lead and the transportation and processing of used batteries were to be regulated under the Canadian Hazardous Products or Transportation of Dangerous Goods Acts, the industry might well be forced out of business. This would result in the same dumping problem that is now being experienced in the United States, as well as a shift of greatly escalated collection and disposal costs from the private to the public sector in response to essential public policy demands.

5. The Battery Industry

The battery industry is Canada's largest user of lead, accounting for about half of total lead consumption. Eleven manufacturers, owned by parent firms in the United States and the United Kingdom but managed by Canadian companies, produced 3 730 760 automotive batteries in 1985; an additional 180 000 automotive batteries were imported in new vehicles. The automotive sector of the industry provided employment for about 2000 workers, and another 400 are engaged in the manufacture of industrial batteries (Dick, 1986).

The industry's consumption of lead in 1985 amounted to 66 900 t (Bigauskas, 1986b), but not all battery manufacturers make the lead oxide needed for the battery paste; much of it is purchased from the chemical industry. This fine oxide powder is the main source of the relatively low atmospheric emissions from battery plants (less than 0.1% of the total amount of lead handled).

(a) Battery Manufacture

Lead that conforms to alloy specifications is purchased in the form of blocks or pigs, mainly from secondary smelters, or as rolled sheet. Three different techniques are used to make the battery grids that are used to form the plates or electrodes (Roork, 1985).

- The pigs are melted in gas-fired or electric melting pots, and the molten lead is poured into water-cooled moulds from a ladle pot. The solidified lead is then ejected from the mould into a trim die, which cuts off the so-called gates--strips or pieces of lead that obstruct the grid openings. These trimmings are returned to the melting pot. The grids are then age-hardened before being pasted with lead oxide.

- Some industrial battery grids are not poured but are pressure-cast from the melt into the moulds, and then trimmed and hardened.

- Some manufacturers use a continuous-feed process in which rolled sheet lead alloy is fed into a punch. Trimmings from the punch are sold back to the secondary smelters, and the grids are fed directly to the pasting machine, as they do not need age-hardening.

The lead oxide paste is the active material of the battery, and different formulations are used for the anodes (positive plates) and cathodes (negative plates). Lead oxide, taken from large storage hoppers, is mixed with sulphuric acid of appropriate concentration and other constituents in a large batch mixer; ventilation is carefully controlled during all handling of the oxide. The paste is then either manually shovelled into the pasting machine or fed from an overhead cone-feeder, and forced under pressure into the spaces in the aged grids as they pass through the machine on a conveyor. The newly formed plates are then partially dried in vented ovens to 7-9% moisture content. The seemingly dry plates are removed from the conveyor, edge-cleaned and stacked for hydrosetting, which involves slow crystal structure and chemical changes that occur over several days during storage. The dustiest part of the process is from conveyor to storage, and effective ventilation is required, first to control the trim dust and later to prevent draughts during storage from mobilizing dust.

The plates are then formed by the imposition of a direct current while they are immersed in a sulphuric acid tank. After forming, the now positive plates are racked, washed and oven-dried; negative plates are immersed in water for some time to prevent further oxidation before drying.

The anodes, cathodes and separators are then hand- or machine-stacked to form elements, and all the positive and negative plate lugs are connected by common straps that are burned on. (In some plants the burning or casting is done before forming the elements.) These burned elements are then boxed, and terminal posts are burned on by adding lead from a burning stick or a small ladle. Dry and wet charged batteries are finished on separate lines; the latter are shipped with acid in the battery containers.

(b) Emissions and Effluents

The primary source of lead emissions to the atmosphere from battery manufacture is particulates from exhaust stacks. Particulates come from in-plant ventilation systems filtered through baghouses. These filters are usually quite efficient, holding as much as 99.9% of the lead. Another significant source arises from transportation, handling and storage of lead oxide, which ideally should be brought directly into ventilated areas of the plant and not unloaded in open yards. In plants that manufacture their own oxide, its production provides another emission source.

Lead-containing effluent, such as wash water or acid from forming tanks, has to be treated and discharged to the sewers in accordance with municipal regulations. Many modern lead-acid battery plants minimize the discharge of lead-contaminated effluent by using closed systems on operations such as washing of paste mixers and pasting machines. The wash water is recycled and re-used (Roork, 1985).

Total atmospheric emissions of lead from battery plants amounted to 2 t in 1984, and aqueous effluents were 4 t (Jaques, 1985).

(c) The Future of the Battery Industry

New grid alloys are responsible for a lower lead content in batteries, but declining use in other market sectors--gasoline additives, solders (especially in food and beverage cans) and paints--means that the proportion of lead used by the battery industry is growing year by year. Actual growth in lead consumption is dependent upon an increasing vehicle fleet. Though lead-acid batteries have found increasing use in electric-powered trucks and delivery vehicles, their power-to-weight ratio is too low for them to be an effective power source for the automobile market. Indeed, current research indicates that the new generation of high energy density batteries that might be successful in this regard is not likely to be lead-based. Such batteries are unlikely to be commercially viable for many years, however, and even then may be too expensive to use only as starter and booster sources. It seems that the next few years will at best show only slow growth for the industry, and that the longer-term future is uncertain.

6. Paints and Coatings

Paints have been used since the earliest times as both decorative and protective finishes on a wide variety of articles and materials (Nriagu, PWR, HWR). The modern industry distinguishes between paints and coatings on the basis of their end use: paints are the trade sales sector of the industry, and have a strong consumer orientation (Cloghesy, 1985). Coatings have an industrial application, such as the protective coating on food cans to prevent reaction between the metal and the contents, corrosion protection on bridges and structural steel and anticorrosive marine life inhibitors for ships. The thickness of some of these highly effective coatings is often only a few thousandths of a centimetre.

Paints are liquid formulations that, when applied to a surface, form protective films that are usually also decorative. Modern paints are formulated from nearly 2000 different raw materials, and are the products of precisely controlled reactions. Some 35 000-40 000 different products are marketed in Canada by 139 manufacturers (Table III.IV). The liquid portion, often called the vehicle, contains both a volatile constituent (either water or organic solvents) and non-volatile film-forming compounds known as binders.

Table III.IV

Location of Paint and Coatings Manufacturing Facilities

Province	No. of facilities
Atlantic Provinces	5
Quebec	36
Ontario	74
Manitoba	3
Alberta	1
Saskatchewan	2
British Columbia	18
Total	139

Source: Cloghesy, 1985

The film former is a resin or lacquer in solvent-based paints, and a latex emulsion in water-based products.

Pigments--usually in powder form--are added for colour and durability, and various other additives impart qualities useful in paint application, such as flow-body or thickness. Some also provide gloss and resistance to mildew after the volatile portion has evaporated, leaving the binder and pigment as a continuous film. In air-drying resin-based paints, coatings and varnishes, film formation (curing) involves chemical linking of the molecules to form larger ones, or polymers; this process occurs as the binder reacts with atmospheric oxygen after the solvent evaporates.

Latex emulsion paints dry by simple evaporation of the water solvent, leaving a continuous film. This is also the case for nitrocellulose and acrylic lacquers.

Brushing, rolling and spraying are the conventional means of application, but industrial coatings are nowadays often formulated to be applied by electrodeposition, a method that allows precise control of uniformity and thickness. The article to be coated is immersed in a tank of paint dispersed in water, and an electric current is applied to it, causing deposition of the paint. Other new processes include electron beam or ultraviolet radiation curing, rather than simple drying to polymerize the film.

Lead compounds have been used as pigments for millennia. One of the commonest of those used in paints--indeed the only major white pigment available to the industry until the introduction of titanium oxide in the late 1940's--was white lead, a basic carbonate $[Pb_3(CO_3)_2(OH)_2]$. This compound was long made by the so-called Dutch process which, in reality, was first described in Greece by Theophrastos in about 300 BC (Partington, 1937).

(a) Paint as an Environmental Hazard*

Flaking leaded paint on interior and exterior walls and woodwork, particularly ill-maintained housing in the central cores of older cities (Lin-Fu, 1972), on children's furniture and on toys has been implicated in childhood lead toxicity, if not directly, then at least by its contribution to the dusts in which children play. Accordingly, several countries have regulated the amount of lead permissible in paints, and prescribed the uses to which paints containing lead pigments may be put. These countries include those of the European Community, Canada and the United States.

The use of lead in paints in Canada is subject to regulations originally promulgated in 1975 (CRC, 1983) under the Hazardous Products Act (RS, 1968-69). These regulations prohibit the use of lead pigments in coatings of children's toys and furniture and in interior consumer paints, and specify that the lead content of such products not exceed 0.5% (as metal) of the total solids in the coating. Specific labelling requirements were also imposed, but lead was not controlled in exterior paints as no documented instance of lead poisoning was identified--even in the United States**. That country had restricted lead in all consumer paints and children's products to 0.5% (expressed as metal) in 1972, and later decreased this amount to 0.06% (Federal Register, 1977). By the time any of these regulations were promulgated, however, the lead content of paints was already falling rapidly, because of the introduction of titanium oxide pigments.

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* Specific environmental problems in hot spots are dealt with in Section VIII.

**At the Commission's Health Workshop, this was questioned; several instances of possible impact due to flaking exterior paint were mentioned in discussion.

Though these regulations restrict the use of lead pigments, lead-based compounds remain widely used as proprietary paint additives, which are added during the manufacturing process, though their benefits may not become evident until the paint is present as finished film. Lead-based wetting or dispersing agents help make a smooth and uniform product. Zinc-based paints are also in use, but these may pose the threat of excessive zinc exposure, also toxic in human subjects.

The trend in recent years towards latex water emulsions, replacing organic solvents even for exterior paints, has been possible only through the use of lead phosphates. Water penetrates the wood and solubilizes its tannins, discolouring the paint film; the lead phosphates stabilize or tie up the tannins, permitting the use of water-based paints.

Lead soap additives--tallates, octoates and naphthates--are used to promote the drying of oleo-resinous coatings. These include both alkyd paints and several industrial coatings, which also contain drying oils.

Despite the Hazardous Products regulations, the largest use of lead by the paints industry is still as pigment, mainly red lead (lead oxide) and lead chromate (Cloghesy, 1985). Lead oxide is mainly used for corrosion protection of bridges and other exterior steel structures; the protective effect is due to surface reactions between the iron or steel and the lead oxide in the film. Gray (1985) estimated the Canadian market consumption of lead chromate pigments in 1984-1985 to be 2300 t, of which 1600 t were used by the paint industry, 600 t by printing ink manufacturers and 100 t by the plastics industry. Chromate pigments are used where durability or gloss is needed, and find application in vehicle paints and for road markings. About 570 t were used in traffic marking paints in 1984.

There are 139 paint and coatings manufacturers located across Canada, though they are concentrated in Ontario and Quebec (Table III.IV). Two of these manufacture lead chromate paints, together employing about 100 people on these product lines. Of total 1984 sales of lead chromate paints of about $16 million, exports accounted for some $10.5 million.

(b) Specific Environmental Concerns

The two types of paint-related concerns are escapes from paint manufacturing plants and the activities of users (Cloghesy, 1985).

Virtually all the 139 paint and coatings manufacturing facilities in Canada are likely to have some lead compounds on the premises, but only those primarily making organic solvent-based paints are likely to have appreciable quantities on hand. Of course, the two manufacturers of lead chromate paints must store quite large quantities of the pigment.

In-plant operations are controlled by provincial occupational health and safety regulations, and their emissions are monitored. Interior dust is carefully controlled, and the inhalation hazard is minimal after the pigments and additives are mixed with the solvents. The only interior airborne particles are likely to be the quite heavy pigment crystals--and of course only in those plants where they are used. A materials balance provided by one manufacturer of lead-based paint showed an overall loss of 0.23% of the input lead. Of this, atmospheric emissions and treated effluent discharges to sewers each accounted for 2.5%. The remaining 95% of the lead loss was in the form of solid waste, and was disposed of to approved landfill sites.

Consumers appear to be more at risk from using paints than from releases from paint manufacturing plants. Sanding of old interior and exterior lead paint before repainting can produce large quantities of lead dust, as does the sanding of bridges (Bonnier, J.-G., 1986; see also Section VIII)*. Lead chromate road paint wears off after a few years, presumably contributing to street dust. Southwood (1983) quoted tests by the Laboratory of the Government Chemist in Britain that showed lead in chromate paint dust to be very insoluble, even in dilute acid (Table III.V). Nevertheless, he recommended that steps be taken to reduce the contribution of road paint to the lead burden of the population, in line with the general view of the Royal Commission in London that all inputs of lead should be reduced or eliminated where practicable. More recently, Morgan (1984) concluded that lead is much less readily absorbed from chromates than from other compounds, though the data examined were more typical of occupational than environmental exposures. Dusts from road paints, however, probably contribute more lead to the environment than does the small amount in road tar (black top) from the use of reprocessed waste oil cake in which the lead is tightly held (Fisher, 1985).

7. The Chemical Industry

The major lead-containing products of the chemical industry are tetraalkyl lead gasoline additives, though the quantities produced are falling annually under federal government regulations, and will continue to do so. Until 1962, the basic carbonate, white lead [$Pb_3(CO_3)_2(OH)_2$], was the next most important product, but the principal product lines are now oxides and chromates.

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* Some local authorities now use zinc-based paints, which have similar anti-corrosive properties. Zinc, however, may also impede the biosynthesis of heme (see Section VII), if it rises above concentration necessary for synthesis.

Table III.V

Lead Analyses of Yellow Road Paint

Sample		Total lead (% by weight)	Soluble lead* (μg/g)
Powdered paint		2.1	20
Lump samples from different sources	1	1.4	150
	2	1.0	250

* Soluble lead was determined by the method specified in the U.K. Toys (Safety) Regulations 1974.

Source: Southwood, 1983

The first step in oxide manufacturing is the slow oxidation of molten lead in a continuous reactor. The molten lead is agitated in an air stream, forming minute droplets that react with the oxygen to form uncalcined litharge or Barton oxide, which still contains a small amount of unoxidized lead. Except for the melting of the lead, this process is operated as a closed circuit, with all dusts being collected through the ventilation system and the oxides collected in vented silos (Figure III.6). The oxides are then loaded into bags or bulkers, which are washed or vacuumed before shipment. Most of the Barton oxide (80%) is used to make battery paste.

For many chemical manufacturing purposes, the remaining lead in the Barton oxide has to be converted to lead oxide, or calcined to oxidize the remaining lead metal. It is again heated in an air stream, and the resulting litharge (PbO) is collected and packed in bulkers, drums or bags. This compound is used to make the wide variety of chemical compounds shown in Table III.VI, again in closed systems vented for dust control.

Some of these compounds are used as stabilizers for polyvinyl chloride (PVC) plastics, which are used as electrical insulation, for plastic piping and ducts, and in many other applications. Light, air and heat cause degradation of the PVC structure, with loss of its properties and formation of hydrogen chloride gas. The lead stabilizers are added to the PVC resin during its manufacture, and need to be handled under properly ventilated conditions until mixed in, when they no longer present a hazard.

Lead oxides are used in many technical glasses where resilience and refractive properties are important. These include optical and flint glasses and photographic and other special lenses. Their ability to absorb radiation has led to the use of lead oxides in colour television tubes to absorb the

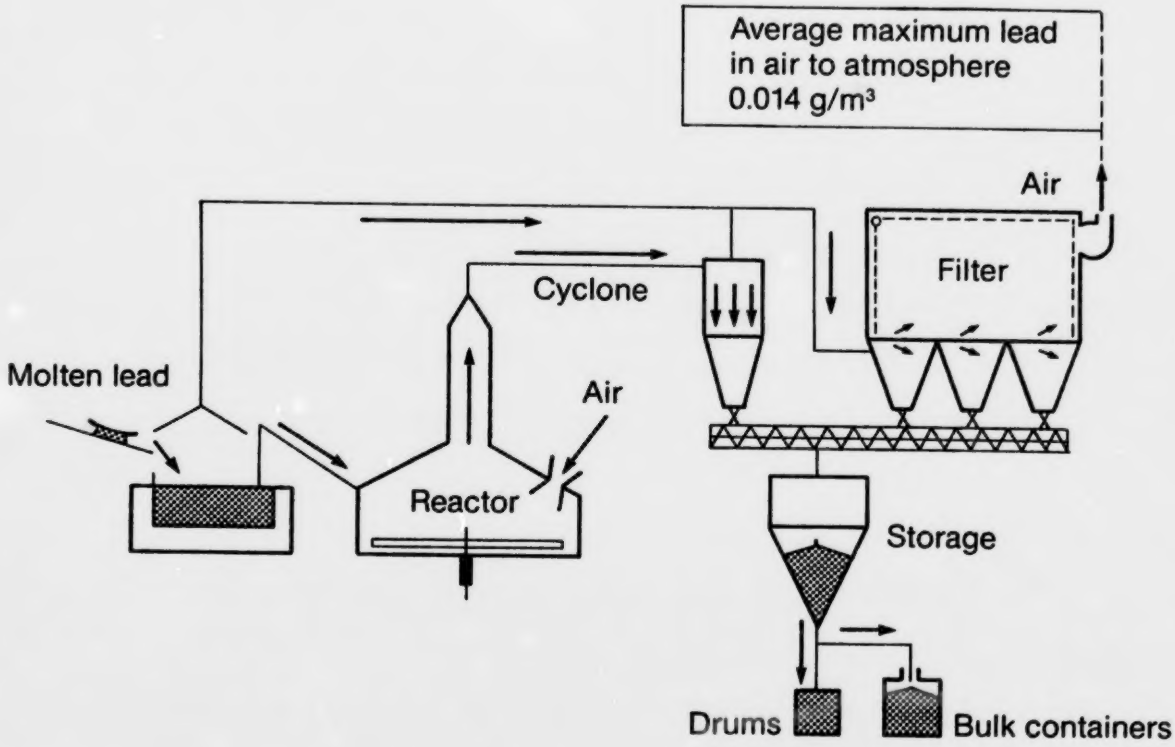
Average maximum lead
in air to atmosphere
0.014 g/m³
Air
Filter
Cyclone
Molten lead
Air
Reactor
Storage
Drums
Bulk containers

Figure III.6. Barton oxide manufacturing.

Source: F. Bonnier, 1985.

Table III.VI

Major Uses of Litharge in the Chemical Industry

Reagent	Product	Use
Sulphuric acid	Lead sulphates: sublimed lead to tetrabasic lead sulphate	Lead stabilizers
Fumaric acid	Tetrabasic lead fumarate	"
Phthalic anhydride	Dibasic lead phthalate	"
Hydrochloric + silica	Lead chlorosilicate	"
Sulphuric acid + silica	Silico sulphates of lead	"
Sulphuric acid + silica	Silico sulphates of lead	Mineral pigments
Phosphorous acid	Dibasic lead phosphate	"
Carbon dioxide + water	White leads	"
Chromic acid	Chromates	"
Acetic acid	Acetates	Other chemicals
Nitric acid	Lead nitrates	"

Source: Bonnier, F., 1985

X-rays produced. Glasses with high lead content are used in the nuclear industry to protect workers against dangerous gamma radiation.

Litharge is also used to make paint driers--the octoate and other soaps described earlier. It also finds applications in the manufacture of mirrors, as well as fillers and polymerizing agents in the rubber industry, and in piezo-electric ceramics, which are used to stabilize frequencies in radio transmitters and receivers. Multi-grade automotive lubricating oils also contain small amounts of organic lead, and lead oxides are among the many substances used in clutch and brake linings.

Tetraethyl lead has been used as an antiknock additive in gasoline since 1922. The steadily falling demand for TEL resulting from regulation has led to a phase-out of production in Canada. Ethyl Canada Ltd. is now the only remaining manufacturer (see Section IV).

In 1984, the industry produced 14 000 t of TEL, equivalent to 9000 t of lead metal. In the last year for which statistics are available (1982), automotive emissions of lead were 7278 t, about 63% of the total atmospheric emissions of lead in Canada (Jaques, 1985).

The manufacture, use and environmental emissions of these compounds are described in Section IV.

8. Solders

About 7500 tonnes of lead are used annually to make 9000 t of various solders. The proportions of lead, tin and other metals used depend on the specific uses for which the solder is required.

Solders are low-melting alloys that will flow into the crystalline structures of other metals and solidify, forming strong alloy-like bonds and leakproof seals. They have long been used to seal food and other cans, to make connections in plumbing and automobile radiators, as automotive body fillers and to make electrical connections, especially in electronic circuitry. Certain applications require high-purity metals, whereas others can use remelted scrap.

Solders are usually made by extrusion from the melt in forms dictated by their use. Thus, plumbing solder is sold in fairly thick rods that are used with externally applied fluxes--usually resinous materials that improve the flow and penetration characteristics. Electronic solders, on the other hand, are extruded around a core of the resin, allowing precise work with a hot tip gun.

Solder scrap and residues are collected and recycled, and old radiators and copper plumbing are similarly handled by the secondary copper industry or brass foundries. Disposal of solders used in many other applications has posed potential environmental and health problems.

Some of the lead emissions from steelworks arise from the recycling of old cars--lead body filler ends up in the steelmaker's flue dusts. These fillers are also being phased out. Lead solders used in plumbing may be leached out, and pose problems in areas with slightly acidic soft water. The use of lead solders in kettles has long been regulated under the Hazardous Products Act (RS, 1968-69).

Requirements for the control of emissions and effluents from solder manufacturing plants are similar to those for secondary smelters. Ventilated hoods, enclosures and baghouses are needed to keep within government-set limits.

9. Cans

About 3.5 billion food cans were used in Canada in 1985. About 1 billion of these cans had lead-soldered side seams. Many foodstuffs can mobilize lead from the solder (Dabeka, HWR), and the cans themselves end up in municipal garbage. Lead limits in certain canned food products are set in regulations under the federal Food and Drug Act: infant formula (0.15 μg/g), fruit juices (0.20 μg/g) and tomato products (0.5-1.5 μg/g).

Soldered food cans are now being phased out, and about 25% of the industry's 1985 production consisted of lead-free welded cans. This type of can is expected to account for 95% by the end of 1986 (Grant, 1985).

A variety of beers and carbonated beverages has been canned in Canada for over 30 years; the regulated limit for lead in beverages is 0.2 μg/g. Production of unsoldered cans with welded or cemented side seams started in 1977, and by 1985 accounted for half the 1.98 billion beverage cans made in that year. The can industry is expecting to complete its conversion to welded two-piece beverage containers made of aluminum or steel by the end of 1986.

The demand for soldered cans used to package other products is also decreasing. Motor oils and other automotive products are now widely packed in plastic containers, and a similar approach is being seriously considered by the paint industry. Many solvents and wood preservatives, however, are likely to continue to be packed in metal containers. The non-food market is currently about 226 million cans annually, with an additional 240 million predominantly welded aerosol containers (Grant, 1985).

10. Miscellaneous Uses of Lead

Some of the more important but minor uses of lead are shown in Table III.VII (Hickson, 1985). Lead sheathing for the protection of telephone and power cables from mechanical damage and corrosion consumes about 2000 t of metal annually. Cables so protected are often laid underground or underwater. The lead is continuously extruded directly onto the cable during manufacture. Lead has been largely replaced by other materials in this application, and the tonnages used are much reduced. Old cables are sold for recycling to secondary lead and copper smelters.

Lead is also used in a number of copper alloys, usually in proportions of about 1-6%. These alloys are wear-resistant and are often used for machined parts and moderate-speed bearings. The manufacturers' scrap is generally recycled. The only possible public exposure to lead may arise from household plumbing fittings made from these alloys. These should now conform to a Canadian Standards Association (CSA) specification dealing with contamination. Only 300 t of lead per year are used in these alloys.

Babbitt and type alloys consume 400 t of lead. Babbitt metals are used for special bearings and bushings, and type metals are used today only for low-volume speciality printing. Both alloys are occasionally used for cast costume jewellery and hot-dip protective coatings.

Semi-finished products include lead pipe, sheet lead, lead roofing, anodes, weights, ammunition and radioisotope containers. Cast lead anodes are often used in the electrolytic recovery of primary metals, and sheet is extensively used as shielding from medical X-ray machines. The heavy density of lead sheet also lends itself to its use as sound-proofing. These products are

Table III.VII

Miscellaneous Uses of Lead

Use	Amount (t)
Cable coverings: undersea and underground cables	2 000
Copper alloys: bronze, brass, etc.	300
Babbitt and type: bearings and speciality printing	400
Semi-finished products: pipe, sheet lead, weights, radioisotope containers	5 500
Ballast, counterweights and shielding: keels, radiation shielding, balance weights	7 000
Total	15 200

Source: Hickson, 1985

usually formed by rolling, stamping or extrusion. Other products include keels for sailboats, counterweights and lead bricks for shielding in nuclear power plants. These uses account for 12 500 t of Canada's annual lead consumption.

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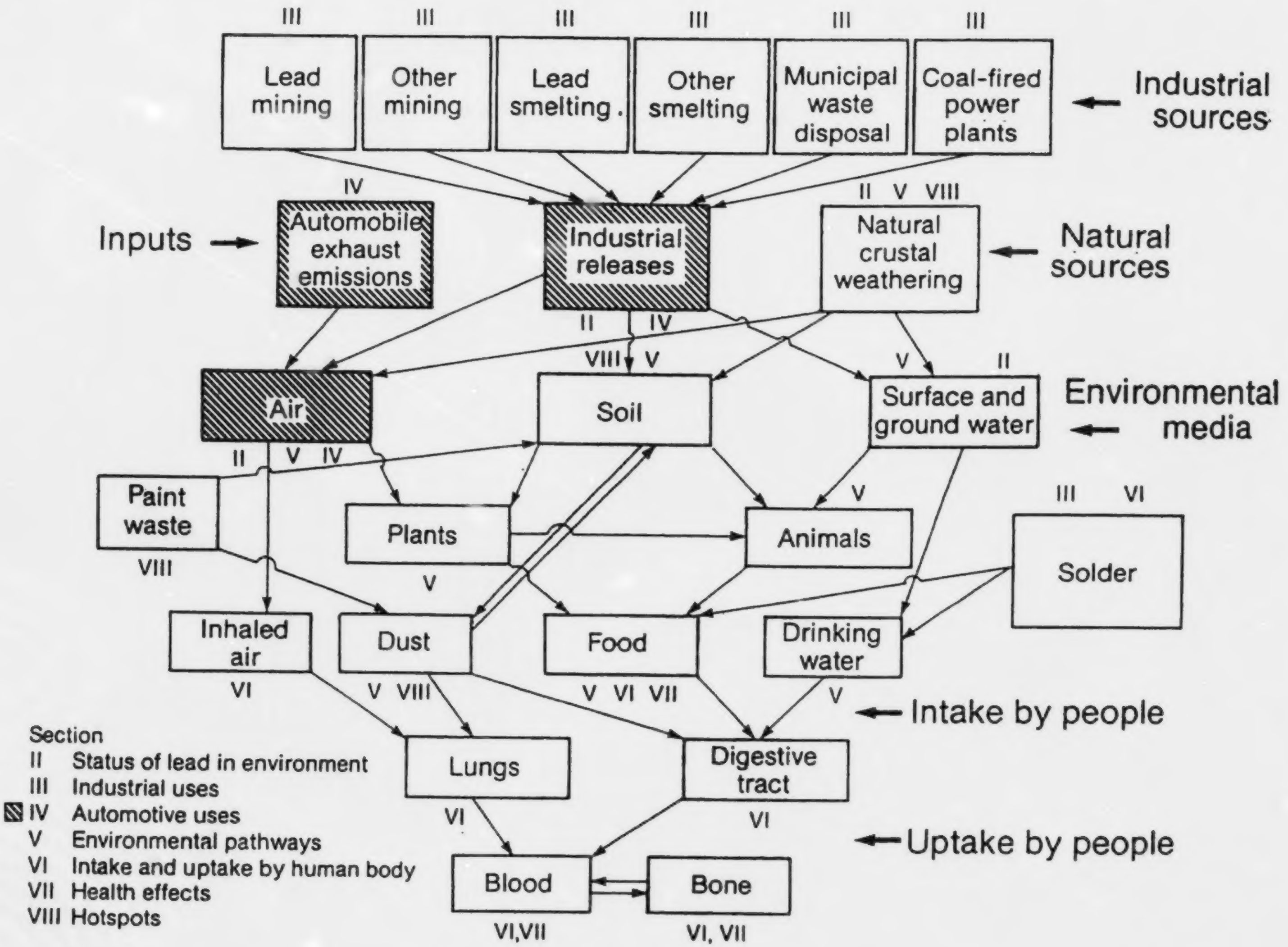
sections (numbered) of text


III III III III III III
Lead mining
Other mining
Lead smelting
Other smelting
Municipal waste disposal
Coal-fired power plants
Industrial sources
IV
Inputs
Automobile exhaust emissions
Industrial releases
II V VIII
Natural crustal weathering
Natural sources
II IV
VIII V
V II
Air
Soil
Surface and ground water
Environmental media
II V IV
Paint waste
VIII
Plants
V
Animals
V
III VI
Solder
Inhaled air
VI
Dust
V VIII
Food
V VI VII
Drinking water
V
Intake by people
Lungs
VI
Digestive tract
VI
Uptake by people
Blood
VI,VII
Bone
VI, VII
Section
II Status of lead in environment
III Industrial uses
▨ IV Automotive uses
V Environmental pathways
VI Intake and uptake by human body
VII Health effects
VIII Hotspots

Area covered in Section IV

IV Use of Lead in Automotive Fuels

1. Why Lead is Used in Motor Gasoline

As shown in Section II, the largest source of environmental lead emissions is still motor gasoline exhaust, though it has been declining. This practice spreads the metal through Canada's populated areas. This source will now be examined in detail (see also the Commission's interim report, "Lead in Gasoline: A Review of the Canadian Policy Issue").

The four-stroke internal combustion engine is the chief source of power that drives automobiles and light-duty trucks. The engine works on the principle that a homogeneous mixture of fuel and air is compressed by a piston before being ignited by an electric spark. The resulting combustion forces the piston outward, and turns the driveshaft. The degree of compression of the fuel and air, normally expressed as the compression ratio of the engine, is a principal determinant of the engine's efficiency. Within certain limits, a higher compression ratio produces greater power per unit mass of fuel consumed. Engine designers therefore strive to develop engines with the highest compression ratio allowed by available fuels.

In the early development of the internal combustion engine, the limitations of fuels restricted the use of high compression ratios. A phenomenon known as knock occurs in the engine when fuel of low octane rating* is compressed and ignited. The knock produced by premature ignition of the fuel reduces the efficiency of the engine, and can also cause major mechanical damage to engine components. The discovery in the 1920's that alkyl lead compounds could reduce engine knock allowed rapid advances in the design and efficiency of automobile engines. In two decades the compression ratio of engines doubled, the power per unit cylinder volume went up by a factor of three and engine efficiency doubled. A more detailed account of these technical questions is offered in Appendix III.

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* For a discussion of the meaning of octane ratings, see Appendix III.

2. The Manufacture of Lead Antiknock Compounds in Canada

Commercial manufacturing of lead antiknock compounds began in the United States in 1923. The product was imported into Canada by Ethyl Canada Inc., which began marketing tetraethyl lead (TEL) in 1926 to Canadian gasoline manufacturers.

In 1956, Ethyl Canada Inc. established a manufacturing facility at Corunna, Ontario, to produce lead antiknock compounds for the Canadian market. A second facility was established by Du Pont Canada Inc. at Maitland, Ontario, in 1965. Declining demand led to the closure of this plant in 1985.

A batch process system is used to manufacture two types of lead antiknock fluid, TEL and tetramethyl lead (TML). The materials required for manufacture include pig lead, ethyl chloride or methyl chloride and ethylene dichloride (produced in Canada), and ethylene dibromide and sodium, which are imported.

In the manufacturing process, molten sodium is combined with molten lead to form an alloy. The highly reactive alloy is then autoclaved with either ethyl chloride or methyl chloride to produce TEL or TML antiknock compound. For TEL the reaction is:

$$4PbNa + 4CH_3CH_2Cl \rightarrow 3Pb + 4NaCl + (CH_3CH_2)_4Pb$$

Ethylene dichloride and ethylene dibromide are added to act as ignition promoters and scavenging agents in the ultimate combustion process in the automotive engine.

The TEL is blended into motor gasoline by the refiner. The amount of TEL added to the blend of gasoline is measured by weight or by flow meter. Concentrated TEL fluid is educted from a storage vessel as the blend of gasoline is produced, and the finished product is usually mixed to ensure a homogeneous blend of components.

The presence of TEL in the motor fuel promotes its more orderly combustion in the engine combustion chamber, with end-gas in the chamber igniting and burning as combustion progresses rather than detonating, and thus causing knock in the engine (see Appendix III).

Though small quantities of TEL (0.06% by mass) produce a substantial increase in octane rating of gasoline, the effect is not linear. In most gasoline components the first gram of fluid produces a much greater percentage of octane increase than does the last. Thus, in the calculation of the amount of TEL to be used, the refiner/blenders have developed very specialized computer-based models to predict the final octane of a blend, and to optimize the blending of components and lead additives.

As long as a market remains for leaded gasoline, the refiner/blender can direct the highest-octane blending components to the lead-free gasoline pool, and upgrade the low-octane components by blending them to the leaded gasoline pool where octane specification is attained by adding TEL.

3. Health Risks in Manufacturing Lead Antiknock Compounds

The risk to health of workers in the lead industry or in the manufacture of lead antiknock compounds is not within the Commission's terms of reference. Nevertheless, early bad experiences of workers both in the antiknock compound industry and in refineries using the product made clear the extremely toxic nature of lead antiknock fluids. Cases of lead poisoning in the industry resulted in the development of precise procedures for the transport, storage and blending of antiknock compounds, and for the handling and disposal of residues from vessels containing leaded gasoline.

4. Lead Emissions from Automobile Fuels in Canada

(a) Combustion of Lead Antiknock Compounds

Lead antiknock compounds are produced in the form of both TML and TEL. For the purpose of simplifying terminology, we will discuss only TEL, more of which is used in the blend.

The TEL antiknock compound contains 394 g of lead in each kilogram of the mixture, loosely called the compound. The percentage composition of TEL compound is shown in Table IV.I.

The antiknock compound in motor gasoline is completely combusted* in the engine cylinder. The scavenging agents, ethylene dibromide and dichloride, which form 37% of the compound by mass, are present to promote the complete combustion of lead compounds. Thus, the lead emitted from the exhaust of vehicles is primarily in the form of inorganic particles (e.g.

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* It is not adequate to substitute "burn" for this technical term.

Table IV.I

Composition of Ethyl Antiknock Compound, TEL

Component	% by mass
Tetraethyl lead	61.48
Ethylene dibromide	17.86
Ethylene dichloride	18.81
Dye, kerosene, antioxidant	1.85
Total	100.00

Source: Ethyl Canada Inc., 1985

PbBrCl), with only small amounts (less than 10% of total emissions) in the form of organolead vapours (EPA, 1986).

Studies in the United States (Pierson and Brachaczek, 1976; EPA, 1986) have shown that these lead emissions are initially very small particles (less than 0.1 μm in diameter) which, when airborne, can remain suspended for up to 30 d and can travel thousands of kilometres. The particles are also susceptible, however, to agglomeration into heavier particles, which settle more rapidly near the source of emission (along highway verges, in most cases).

In addition to small particulate lead emissions, there is some lead that agglomerates and remains on the surfaces of the engine or exhaust system, and still other lead that is flushed into the oil reservoir. These accumulations may later be released to atmosphere or soil, when the oil or scrap exhaust systems are destroyed. Table IV.II shows an estimate (EPA, 1986) of the forms assumed by this combusted lead.

(b) Lead Emissions from Gasoline in Storage

The vapours from gasoline in storage or transit contain small amounts of TEL in equilibrium. These vapours of lead are in organic form and are photoreactive. It is estimated that the atmospheric half-life of organic lead vapour is less than 12 h (Neilsen, 1982). These emissions are hence of greater concern in the industrial setting than in the general environment.

(c) Emissions by Misfuelling

The term misfuelling refers to the practice of drivers who use leaded fuel in a vehicle designed to operate on unleaded fuel. Though there is no precise figure of the actual extent of misfuelling, there is sub-

Table IV.II

Forms of Lead Derived from Motor Fuel

Form of lead emitted	% of lead in fuel
Lead aerosol MMD* less than 0.25 μm	35
Lead particle MMD greater than 10 μm	40
Retained on engine and exhaust surfaces	15
Accumulated in engine oil	10

* MMD = mass median diameter.

Source: EPA, 1986

stantial evidence that the practice is fairly common in Canada*. Surveys of retail gasoline outlets have revealed small-diameter (unleaded fuel) fuelling nozzles on leaded fuel pumps, unleaded vehicles with no restriction in the filling pipe and vehicles with the catalytic converter removed (Optima, 1985).

In the calculation below of future emissions, it has been assumed that misfuelling will be eliminated by provincial action in future years, and that this will not increase emissions above the calculated levels. In its interim report, "Lead in Gasoline: A Review of the Canadian Policy Issue", the Commission recommended action by Governments to overcome this practice.

(d) Emissions from Marine and Aircraft Engines

Emissions from marine fuel sales are included in estimates given below to 1984, but are not projected for subsequent years. These emissions are generally in non-urban areas, and much of the lead emitted enters the water.

Similarly, exhaust emissions from aircraft burning leaded gasoline are so widely dispersed (1.7% of total lead emitted; Environment Canada, 1984) that no accurate account of the effect on the environment can be made.

(e) Emissions from Disposal of Waste Oil

Oil drained from the crankcases of engines enters the environment in a number of ways. Some used oil is sold to oil reprocessors (approximately 18%), whereas other amounts find their way into fuel oil and are burned in heating plants. The used oils that are burned create a point source of lead in the area (see Section III).

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* A survey done for Environmental Canada in 1985 indicated that 12% of individual sales involved misfuelling.

5. Future Automotive Exhaust Emissions in Canada

The Inventory Management Division of the Environmental Protection Service of Environment Canada provided the Commission with estimates of automotive lead emissions in Canada for the period 1933-2003. These are reproduced in Appendix IV (Vena, 1985).

For the period 1933-1978, the figures are based on the consumption of lead antiknock fluids used in fuel blended in Canada. The volumes of antiknock fluid were supplied by Ethyl Canada Inc. For the period 1978-1983, the amount of lead antiknock fluid added to gasoline in Canada was reported by the refiners and blenders. In all cases, it is assumed that all lead blended in gasoline in a year is emitted in that year. Furthermore, it is assumed that 75% of the lead used in a year is emitted in the exhaust of the vehicle.

For the period 1984-2003, the estimates are based on a combination of studies and projections. The major sources of information are (i) the National Energy Board projections of energy use, and (ii) Du Pont of Canada's Escon model prediction of gasoline demand. This model considers the mix of vehicles, fuel efficiency and average vehicle mileage. An integral part of the forecast for future years is the implementation of recent regulations affecting future lead content of fuel and mix of vehicles types. It is therefore assumed that all leaded fuel will contain a maximum of 0.29 g/L of lead from January 1, 1987, and that all new light-duty vehicles sold in Canada after September, 1987 (the 1988 models), will be equipped with catalytic converters, and will require unleaded fuel.

Submissions to the Commission by Environment Canada and by PACE (Petroleum Association for Conservation of the Canadian Environment) give both historical and forecast data for lead emissions from vehicles (see Appendix

IV). Though the predictions from the two sources are not identical, the differences are due to the assumed rates at which lead-tolerant vehicles are retired after 1987. The PACE forecast shows a more rapid decline of emissions after 1987. However, the difference is only 5% in the forecast for 1993, and is not significant in the conclusions to be drawn from the data. The historical and forecast data based on current (i.e. 1986) regulations are shown in Figure IV.1.

Automotive emissions of lead increased from 2600 t in 1948 to a peak of 14 360 t in 1973. The introduction of lead-free gasoline in 1972, and subsequent restrictions on the use of lead in leaded gasoline, combined to produce a decline in the emissions of lead to the atmosphere. By 1984, emissions were reduced to slightly less than half the 1973 peak.

A further reduction in the permissible lead in leaded gasoline will be implemented in 1987 by regulations under the Clean Air Act, 1971. It is predicted that lead emissions will be more than halved again, bringing the emissions level in 1987 to 2750 t. In historical terms, this will be equivalent to the emissions in 1947. After January 1, 1987, the amount of lead added to gasoline will be reduced by 33% to 0.29 g/L, and the population of vehicles that must use unleaded fuel will increase. Emissions of lead are forecast to decline at a rate of approximately 11% per year thereafter, as long as the 1987 regulations remain in force.

A further reduction in the allowable concentration of lead in leaded gasoline to 0.026 g/L (the current US standard) would reduce annual emissions of lead from automobiles to 141 t from December 31, 1992. This level would be less than 1% of the emissions of 1973, and the majority of vehicles using

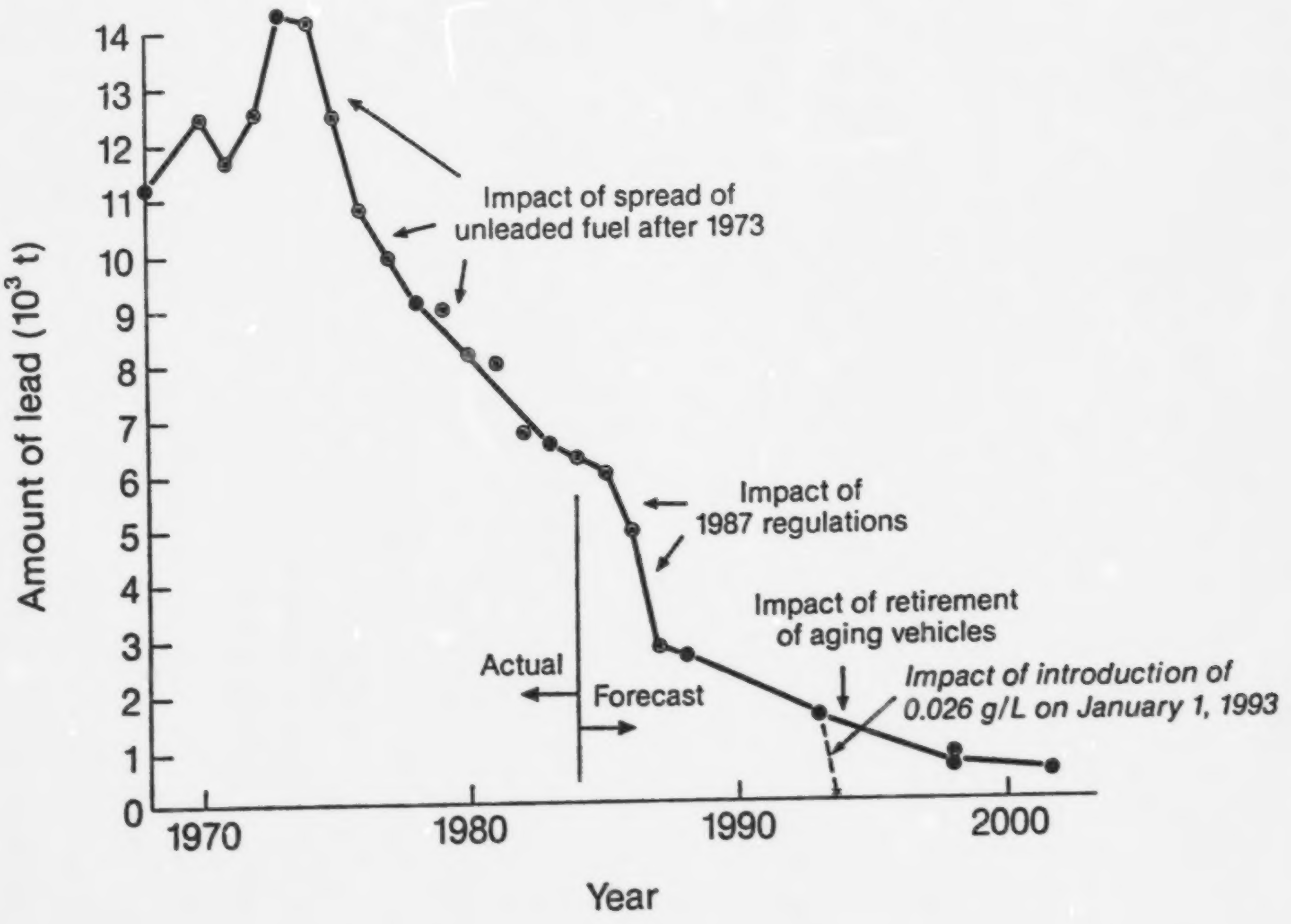

Figure IV.1. Automotive lead emissions in Canada, 1968-2000.

Source: Environment Canada (●) and PACE (⊙). See Appendix IV.

leaded fuel would be in rural or remote areas (see impact of proposed 0.026 g/L regulation in Figure IV.1). The Government of Canada has not yet, however, announced the actual level (which may be zero) to apply from this date.

6. Regulations on the Use of Lead Compounds in Motor Fuels

(a) Canadian Regulations

Unleaded gasoline was introduced in Canada in 1972 to allow the use of catalytic converters on new vehicles. The purpose was to control emissions of hydrocarbons, carbon monoxide and nitrogen oxides, and not lead.

Under the Clean Air Act, regulations "may be prescribed in regard to the concentration of elements or additives for fuels which result in significant contribution to air pollution". The Leaded Gasoline Regulations were made by Order in Council. Under these regulations the maximum allowable concentration of lead in gasoline was established at 0.77 g/L (3.5 g per imperial gallon) (Canada Gazette, 1974).

This regulation remains in effect in 1986, though the producers of leaded gasoline do not blend to the maximum allowable content of lead. The consumption of lead antiknock compound indicates that leaded gasoline in Canada contained 0.45 g/L on average in 1984.

On May 3, 1984, the Leaded Gasoline Regulations of the Clean Air Act were amended so that "the maximum concentration of lead in gasoline to which lead has been added during production--shall not exceed, (a) prior to January 1, 1987, 0.77 g/L, and (b) on or after January 1, 1987, 0.29 g/L" (Canada Gazette, 1984).

Proposed regulations for new cars under the Canada Motor Vehicle Safety Act were announced by the Ministers of Transport and Environment on March 6, 1985: "the adoption of tighter new vehicle emission control standards to reduce harmful air pollution. The standards to be adopted will be equivalent to those currently in effect for cars and light trucks in the United States. They will take effect on September 1, 1987" (Environment Canada, 1985). The new standards will require that 1988 model light-duty vehicles be equipped with exhaust converters, thus making unleaded fuel mandatory for these vehicles. This regulation will reduce still further the amount of leaded gasoline sold in Canada.

On March 25, 1986, the Minister of the Environment announced "plans to virtually remove lead from the nation's fuel supplies by December 31, 1992". Though no specific regulation was proposed, an official of Environment Canada indicated that "at the very least, the government will adopt the US standard implemented in 1986 of 0.026 grams per litre". This is consistent with the Commission's recommendation in its interim report (p. 50), though a date of 1990 was believed to have been feasible.

(b) United States Regulations

In the United States, lead in gasoline was regulated from January 1, 1975. These regulations were drafted to control the average lead content of the total pool of gasoline to a maximum of 0.5 g per US gallon (0.13 g/L) by January, 1979. Regulations allowed refiners who did not need their full quota of lead antiknock rights to sell these rights to other refiners. The smaller refiners who did not have high octane producing processes could thus purchase the right to add more lead antiknock compound to their gasoline. This right expires in 1988.

In 1982, EPA adopted a new approach aimed at regulating the lead in leaded gasoline rather than the total pool. A new standard of 1.1 g per leaded US gallon (0.29 g/L) was established. This was roughly equivalent to the old 0.5 g per total pool gallon.

On March 7, 1985, EPA promulgated "a low lead standard of 0.10 grams per leaded US gallon (0.026 g/L), effective January 1, 1986 and an interim standard of 0.50 grams per US gallon (0.13 g/L) effective July 1, 1985" (EPA, 1985).

The order requested comments on "a ban on the use of leaded gasoline by about 1995". These comments were to identify "whether alternatives exist to replace lead as a valve lubricant, and the number of vehicles that would require lead for this purpose in both the short and long term". The agency decided to solicit additional comments on the subject.

(c) European, Pacific and Latin American Standards

Current European regulations on lead content in fuel apply to the total pool of gasoline. European Community (CEC) Member States have not established a uniform standard for the lead content of gasoline, but have a maximum of 0.4 g/L. The CEC has, however, established guidelines stipulating that:

- starting on January 1, 1986, any country on its own initiative can market lead-free gasoline. (The Federal Republic of Germany has 1300 stations supplying lead-free gasoline. Certain other countries have it available on a more limited basis)

- on July 1, 1989, lead-free gasoline must be available everywhere in the territory of member countries

- on July 1, 1989, a new guideline limits the maximum level of lead in leaded gasoline to 0.15 g/L (Valais and Bonnifay, 1986)

In other European countries, current regulations allow total pool lead contents ranging from 0.15 g/L in Austria, Norway, Sweden and Switzerland to 0.64 g/L in Portugal. It is expected that all countries will fall more in line with the current EEC guidelines within the next 5 years.

(d) New Zealand and Australian Standards

New Zealand allows lead at 0.84 g/L in the total pool of gasoline at present, but is expected to reduce the permitted level to 0.40 g/L by 1987. Australian regulations vary by state, and range from 0.84 to 0.30 g/L in the total pool. Unleaded gasoline is being introduced in some states.

(e) Comparison of Canada, US and Europe

Table IV.III tabulates regulations from all available countries. A comparison of the standards proposed in Canada and the US with EEC countries on a total pool basis is shown in Table IV.IV.

7. Lead Compounds as Valve Lubricants

Some internal combustion engines are designed specifically to use leaded fuel. The lead in the fuel acts as a lubricant to reduce wear between the valve and the valve seat. Unleaded fuel in these engines can produce valve recession, or rapid wear of the valve seat, culminating in loss of compression and engine efficiency.

Table IV.III

International Lead Limits

Area	1984 level (g/L)	Proposed lead reduction Level (g/L)	Proposed lead reduction Date	Proposed unleaded phase-in
CEC				
Denmark	0.15	-		1987
Germany	0.15	0.15		1987
Netherlands	0.40	0.15	1986	1987
UK	0.40	0.15	1986	1989
Belgium	0.40	0.15	1987	1989
Ireland	0.40	0.15	1989	1989
France	0.40	0.15	1989	1989
Italy	0.40	0.15	1989	1989
Greece	0.40	0.15	1989	1989
Athens	0.15	-		
Other European countries				
Austria	0.15	-		1987
Norway	0.15	-		1987
Sweden	0.15	-		1987
Switzerland	0.15	-		1986
East Germany	0.40	0.15	1989	1987
Czechoslovakia	0.40	0.15	1989	1987
Finland	0.40	0.15	1985	1989
Portugal	0.64	0.40	1986	1989
Spain	0.60	0.40	1986	1989
Yugoslavia	0.60	0.40	1989	1989
Asia Pacific/Latin America				
Taiwan	0.30	-		1985
Hong Kong	0.40	0.15	1987	-
New Zealand	0.84	0.40	1987	1987
Singapore	0.40	0.15	1989	1989
Venezuela	0.84	0.29	1987	-
South Africa	0.84	0.40	1986	1989
Malaysia	0.84	0.40	1989	-
Argentina	0.84	0.40	1988	-
Australia				
Victoria	0.30			
New South Wales	0.40	(Australian jurisdictions are moving towards use of unleaded fuel)		
South Australia	0.65			
Western Australia, Queensland, Tasmania and Northern Territory	0.84			

Sources: Ethyl Canada Inc., 1985; PACE, 1985; EMR, 1986

Table IV.IV

Proposed Total Pool Lead Content, 1987

Country	Maximum pool lead level (g/L)
Canada	0.15
US	0.015
Denmark, Germany, Netherlands, UK	0.15
Other EEC countries	0.40

Source: Keough, 1986

Such engines are, in general, those used in farm vehicles, boats or ships, two-stroke outboard units, aircraft, heavy-duty trucks and construction equipment, as well as older and vintage automobiles.

In the United States, EPA considered the new maximum level of 0.026 g/L for leaded gasoline to be adequate to protect the valves of engines requiring lead lubrication. However, there has recently been some disagreement as to the actual lead content required to protect engines, particularly under the high speed and high load conditions often encountered in agricultural equipment. EPA is undertaking research to establish this level. The complete elimination of lead from gasoline will require special consideration for engines needing valve lubrication.

8. The Search for Octane in Motor Gasoline: Additives

(a) Lead Antiknock Compounds

As indicated earlier, the octane ratings of motor gasoline have been maintained at high levels by the use of TEL additives, but these are now being phased out. In the Commission's interim and supplementary reports, it was indicated (i) that it was thought unrealistic to propose a reduction in octane requirements, given the engine efficiency and fuel economy now required in motor vehicles, and (ii) that alternative sources would accordingly be required. The task is formidable, because TEL is an excellent octane source. The susceptibility of various gasoline fractions to improvement in octane by the use of TEL is shown in Table IV.V.

Today, as in 1923, there is no octane-improver additive or process that competes economically with lead antiknock compounds. There are substantial cost and energy penalties to be paid to maintain octane levels. The alter-

Table IV.V

Octane Response of Gasoline Components to Tetraethyl Lead at a Concentration of 0.8 g/L*

Component	Research octane no. (RON)	
	Clear	Leaded
Crude naphtha distillate	70	89
Catalytic cracked gasoline	89	97
Alkylate	94	102
Butane	94	103
Reformate	97	103

* Current lead addition rate (average for industry) is only 0.45 g/L.

Source: PACE, 1985

natives are reviewed briefly below. A more complete treatment is given in the Commission's supplementary report "Alternatives to Lead in Gasoline".

(b) Methyl Cyclopentadienyl Manganese Tricarbonyl (MMT)

MMT produces a higher octane rating in a blend of gasoline, yet is compatible with the catalyst present in the catalytic converter of an unleaded-fuel vehicle. The amount of MMT approved by the Canadian General Standards Board for use in gasoline is only 0.018 g/L, but its impact on octane is very significantly positive. In general, the cost of producing octane by the use of MMT is twice the cost of using lead compounds, but only half the cost of the best processing alternative.

The Commission considers that the additional public exposure to manganese that will arise from general use of MMT is readily tolerable. Manganese is an abundant element in nature, and we are already exposed to it at levels far exceeding those that will arise from the expanded use of MMT (see the Commission's supplementary report, "Alternatives to Lead in Gasoline").

(c) Methanol, Ethanol and Methyl Tertiary Butyl Ether (MTBE)

Alcohols have long been used as motor fuels or as fuel performance boosters. They are commonly used in countries that have a shortage of petroleum hydrocarbons. The use of alcohols in motor fuels in Europe and South America, particularly in Brazil, is very well developed, and alcohols can be used effectively in certain economic or political situations.

In North America, methanol and ethanol have received renewed attention because of their potential as substitutes for petroleum fuel, and because of their relatively high octane ratings as substitutes for high-cost petroleum reformates.

Though alcohols and ethers have attractive qualities as substitutes for high-octane petroleum fractions, they suffer from the disadvantage of having lower specific heats of combustion than does gasoline. Alcohols also have solubility properties that complicate their use in gasoline blends.

Methanol is used to a limited extent as a gasoline component in Canada. Its major advantage is the abundant potential source of its raw material, natural gas. It has satisfactory combustion qualities and an excellent octane rating, but it can be difficult to handle in the normal gasoline distribution system. Methanol can also create vapour pressure problems that require removal of more valuable butane components from gasoline in summer months.

Methanol is currently used in gasoline blends in Saskatchewan, Alberta, British Columbia and Ontario, and is also used in significant volume in the United States. In all cases, the methanol is blended with a higher alcohol such as ethanol, isopropanol or isobutanol to improve its ability to remain in solution with the gasoline hydrocarbons.

Neat methanol is considered by some automobile manufacturers to be the preferred method of using alcohol as a fuel. This requires a specific engine design to optimize the use of the fuel.

Environmentally, the use of methanol in automobiles leads to higher production of aldehydes in the exhaust, but the amounts are extremely small. They are quite efficiently removed by the catalysts of the exhaust system, so that the possibility of adverse health effects appears to be remote.

Ethanol has been widely used as a gasoline component in North America, particularly in the US mid-west. It has been used widely as a gasoline component in the US mid-west where special state tax treatment has been allowed.

It is also used to some extent in Canada (Manitoba), and as a cosolvent with methanol in gasoline in Saskatchewan, Alberta and British Columbia, in order to overcome water problems.

Ethanol used in North America for motor fuel is produced from fermentation of agricultural crops, and is not thought likely to be a competitive source of significant quantities of motor gasoline blending component.

Ethanol has an advantage over methanol in being more stable in a mixture with hydrocarbons, and in being less difficult to handle in the normal gasoline distribution systems. In addition, it does not increase the vapour pressure of gasoline to the same extent as methanol, and does not, therefore, suffer from the cost penalty associated with a reduction of butane in the gasoline blend.

MTBE is used in significant quantities in Europe and the United States as a high-octane blending component of gasoline. It is permitted in gasoline blends in the United States to a concentration of 11% by volume (EPA, 1979, 1981), under standard under the US Clean Air Act. It is expected that a similar regulation will apply in Canada. This subject is currently being reviewed by the Canadian General Standards Board.

MTBE is manufactured by combining isobutylene and methanol into a high-octane compound that exhibits characteristics very similar to those of the hydrocarbon fuels. It is water-tolerant in the distribution system, and presents no problems of stability, volatility or corrosion.

In the United States, MTBE production capacity is increasing because of a current surplus of methanol and a highly concentrated source of isobutylene from the chemical crackers on the Gulf Coast of Texas. The new EPA regulations on reduction of lead compounds in gasoline have stimulated interest in MTBE, and the price has moved up in response to increased demand for high-octane blending components.

Other MTBE plants are under design or evaluation in the US, Saudi Arabia, Ireland, Scotland, Canada and western Europe. European and US production is approximately 2000 kt/a, with more than 50% manufactured in the US.

In Canada, MTBE is an attractive blending component for gasoline because of the abundant potential supply of methanol. The major problem is to establish an equally abundant source of isobutylene, because the chemical industry in Canada is not concentrated in one section of the country.

It is quite probable that MTBE will be used to some extent in this country after 1987, when the more restrictive regulations on lead come into effect. Whether or not MTBE will be produced in Canada depends upon the economic evaluation versus other processing alternatives.

(d) Other Blending Components: Benzene, Toluene, Xylene

The so-called B-T-X group of aromatic hydrocarbons--benzene, toluene, xylene--contains highly valued components for gasoline blending, with high octane ratings and good blending qualities. The components are produced in the normal processing of crude oil from cracking and reforming processes.

Though these aromatics are very desirable gasoline components, they are also valued as materials for the manufacture of a wide range of chemical compounds. Thus, the B-T-X components are often extracted from the motor gasoline inventory to be used as chemical feedstocks. As lead compounds are reduced in the motor gasoline pool, however, the value of B-T-X compounds will increase. This in turn will have an impact on the price of chemicals made from B-T-X.

Many aromatic hydrocarbons are toxic, and some, notably benzene, are carcinogenic in humans and other animals. The increased use of aromatics as octane improvers for motor gasoline raises concern about the health effects of the additional emissions from automobiles. Though this hazard is difficult to assess, it seems prudent to minimize the presence of aromatic compounds in gasoline (see the Commission's supplementary report). The European Economic Community has limited the use of benzene to 5%. Canada and the US have not so far followed suit.

9. The Search for Octane: Refining Processes for Motor Fuels

In the early 1920's, when the automobile was coming into widespread use in North America, petroleum refiners were searching for process technologies that would convert more heavy hydrocarbons into gasoline components, and at the same time produce branched-chain molecules that exhibit higher octane characteristics. This search for both volume and octane increase continued through the 1960's. At present, with gasoline sales contracting, volume is not an objective, but the search for octane continues.

The sequence of refining process development is roughly as shown below:

1900-1915	Simple distillation
1925-present	Thermal cracking and coking
1945-present	Catalytic cracking
1945-present	Alkylation and catalytic polymerization
1950-present	Naphtha reforming
1960-present	Hydrocracking
1970-present	Isomerization

A typical flow diagram for a Canadian refinery is shown in Figure IV.2.

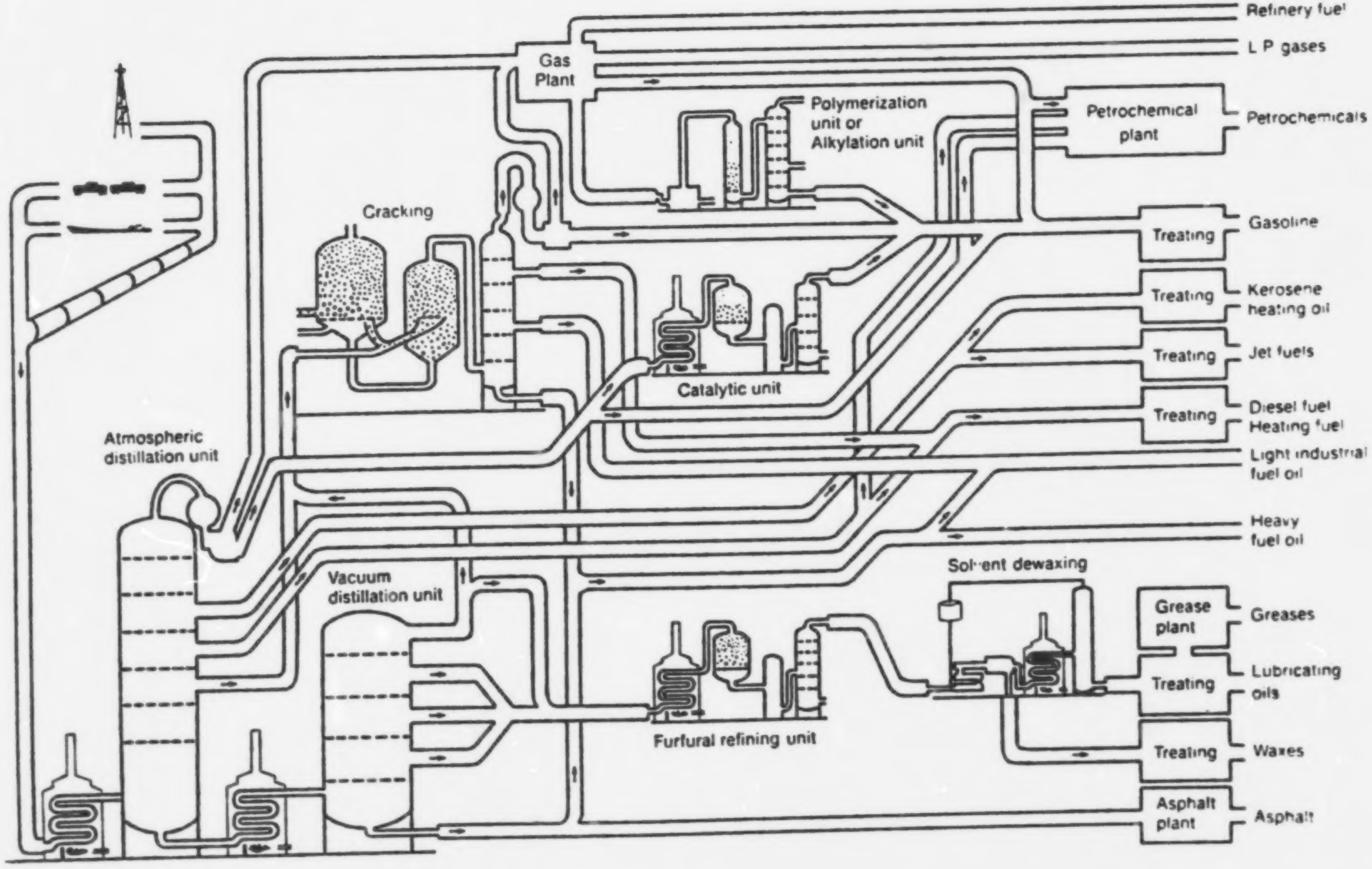
Refinery fuel
L P gases
Gas Plant
Polymerization unit or Alkylation unit
Petrochemical plant
Petrochemicals
Cracking
Treating
Gasoline
Treating
Kerosene heating oil
Treating
Jet fuels
Catalytic unit
Treating
Diesel fuel Heating fuel
Atmospheric distillation unit
Light industrial fuel oil
Heavy fuel oil
Vacuum distillation unit
Solvent dewaxing
Grease plant
Greases
Treating
Lubricating oils
Furfural refining unit
Treating
Waxes
Asphalt plant
Asphalt

Figure IV.2. Flow diagram for a typical Canadian refinery

Source: PACE. 1985

(a) Fluid Catalytic Cracking (FCC), Alkylation and Polymerization

FCC is the workhorse process of modern refineries. It converts heavy oil streams into gasoline and light diesel. In the cracking process, by-product butylenes and propylenes are also produced, and are further processed into high-octane gasoline components in other processing units.

The gasoline produced directly from the FCC unit has a research octane number (RON) rating in the high 80's. It is therefore a good quality blending material, but not good enough to meet market octane demand without some octane improver. Fortunately, the FCC unit also produces butylenes and propylenes that can be alkylated in a catalytic process using hydrofluoric or sulphuric acid to produce motor gasoline alkylate. This component has a high octane rating, and is an excellent gasoline component at today's octane levels.

In some refineries, catalytic polymerization is used in place of alkylation to produce high-octane blend stock from propylene.

(b) Hydrocracking

Hydrocracking is, in a sense, the opposite of fluidic catalytic cracking, in that its objective is to produce molecules that are straight-chain rather than branched, and saturated with hydrogen rather than unsaturated. Hydrocracking converts olefinic diesel and heating oils, in the presence of hydrogen, into naphthas and light distillates for use in motor gasoline, jet fuel and high-quality diesel fuel. Hydrocracking is important as a potential producer of molecules that can be catalytically reformed into high-octane gasoline components. Because it consumes hydrogen to saturate the cracked molecules, hydrocracking produces an increase in the volume of product.

(c) Catalytic Reforming

In a regime of declining use of tetraethyl antiknock fluid, the catalytic reformer is the refiners' critical process unit. Catalytic reforming is a fixed bed process using a noble metal catalyst (usually platinum) to change the molecular structure of naphtha molecules (which contain chains of 7 or 8 carbon atoms, C_7 or C_8 molecules) that are the major components of motor gasoline.

The catalytic reforming process prefers naphtha molecules having only single carbon-carbon bonds as feedstock, and produces a variety of molecular structures from them that depend upon operating conditions. The four major reactions that take place in the reformer are aromatization, isomerization, cyclization and hydrocracking. The straight-chain naphtha fed to the catalytic reformer emerges as a blend of aromatic and branched-chain hydrocarbons that have much higher octane quality than does the feedstock. Because of the complex nature of the cracking and reforming reactions, this process produces surplus quantities of hydrogen and a very significant shrinkage in liquid volume of product.

The ability of a refiner to respond to the production of constant octane motor gasoline, with declining concentration of lead, depends primarily on the capacity of the catalytic reforming process. If a refiner does not have surplus capacity to be used for additional octane quality production, then he must construct new capacity, or purchase high-quality blending stocks such as the alcohols or MTBE discussed earlier.

Catalytic reforming also presents another problem for the refiner at high-capacity operation. The reformer is not effective in changing the structure of C_5-C_6 molecules. As the refiner increases production of high-octane reformate, he also accumulates a surplus of low-octane (in the order of 70 RON), light (C_5-C_6) naphthas, which cannot be used in the gasoline pool without some form of octane improvement.

In such a case, the refiner would have to consider further investment in a process to isomerize the surplus light naphtha to improve octane for blending into the unleaded gasoline.

(d) Isomerization

The octane rating of straight-chain hydrocarbon molecules, especially those with 5 or 6 carbon atoms (C_5-C_6), can be upgraded by a process that restructures the molecule into an isomer (see Appendix IX).

To date, isomerization of low-boiling hydrocarbons in Canada has been dedicated to the production of chemical products, as dictated by prices. However, as the need for additional sources of octane increases, isomerization for motor fuels will become attractive. Isomerization process units can be constructed for single-pass operation, which will improve quality by 10 octanes (RON), or recycle operations that will add 20 octanes (RON). A modest supply of hydrogen is required for the isomerization process.

10. What Will be the Cost of Replacing Lead Additives in Gasoline?

The reduction in the use of lead antiknock compounds has a direct impact on the lead industry, a profound impact on the antiknock producers and significant effect on the facilities and blending components used in the petroleum refining industry. Only the petroleum refining industry will be discussed here.

The Canadian petroleum industry, through PACE, has provided the Commission with an assessment of the cost impact on the industry of the planned reduction of lead in gasoline.

Gasoline consumption in Canada in 1984 was 31.8×10^9 L, and is forecast to decline to 28.6×10^9 L by 1993. The pool octane (i.e. the octane rating of both leaded and unleaded grades) in 1985 was 89.2 Road Octane Index (R + M)/2. It is expected that this quality will be maintained as lead is reduced (see Appendix III).

The use of lead compounds in 1985 contributed 4.3 octanes to the pool octane. As this component is removed, the refiners are faced with either major expansion of facilities to produce octane, or major expenditures for alternative blending components, all of which are much more expensive than alkyl lead compounds.

The components and processing options for octane improvement vary widely in cost, as shown in Table IV.VI. Costs are expressed in cents per octane-barrel, which is standard industry usage. Cents per octane-barrel means the cost to increase the octane rating of one barrel (159 L) of gasoline by one octane number.

Refiners will choose a variety of methods to replace leaded octane quality, according to the configuration of their refineries, the cost and availability of blending components and their strategic decisions on markets to be served. The lead phase-down will give rise to a variety of predictable changes in the industry:

- operating costs of refineries will increase to produce higher octane components from existing facilities

Table IV.VI

Typical Cost Per Octane-barrel Improvement

Component	Cost per octane-barrel (cents)
TEL at 0.29 g/L	6***
MMT at 0.016 g/L	15
Reforming high severity	30
B-T-X aromatics	42-92*
MTBE	40-90*
Isomerization of light naphtha	60-80**

* Aromatics and MTBE are very sensitive to chemical industry demand.

** Isomerization is sensitive to capital cost and capacity.

***The price of TEL has almost doubled recently (summer 1986) as a result of sales pressure.

Source: PACE, 1985

- the consumption of crude oil will increase with the increased severity of refining operations
- costs of process chemicals and catalysts will increase as more severe operations are required to produce higher octane
- major investments in facilities will be required to upgrade components that are currently acceptable only for leaded grades of gasoline
- purchases of high-octane blending components, such as alcohols and aromatics, will be needed

The cost of improving and operating refining facilities, as estimated by PACE, is shown in Table IV.VII. Capital and operating costs are expressed in constant 1983 dollars, and include depreciation charges and return on capital charges for new facilities. These charges will flow through to the retail price of fuel, and will result, in time, in higher cost to the consumer.

The programmed reduction of lead in gasoline to 0.29 g/L in 1987 will create an octane shortfall that will be met with a combination of activities by refiners, including some or all of the following:

- increased capacity and/or severity of naphtha-reforming operations
- increased use of catalysts in catalytic crackers, hydrogenation units, alkylation units and reformers
- increased production or addition of benzene, toluene and xylene in gasoline
- increased use of alcohol and ether blending components
- construction of new facilities such as isomerization units, additive blending facilities and other octane improvement processes

Table IV.VII

Estimated Petroleum Industry Costs of Lead Phase-down

		Cost (millions of constant 1983 dollars)	
Case	Regulation	Additional capital	Additional annual refining cost
Base case	0.29 g/L lead in 1987 and light-duty vehicle regulations; 90/10 mix unleaded/lead fuel	500	275
Future reduction	0.29 g/L lead in 1987, light-duty vehicle regulations; 0.026 g/L lead after 1990	800	445

Note: All cases assume continued use of MMT.

Source: PACE, 1985

- closure of small refineries that cannot be upgraded economically
- imports of high-quality gasoline or additives

The data supplied to the Commission by PACE have been evaluated by Data Resources of Canada (DRI, 1985), as regards their impact on the economy of Canada and effect on contingent industries. However, the Commission believes that the increased costs due to the removal of lead are likely to be masked by other price effects on gasoline, such as changes in taxes, crude oil prices and competitive market forces, all of which cause much larger variations in the price of gasoline than will the removal of lead.

In predicting the cost flow-through to the consumer, DRI assumed (on our advice) that traditional industry pricing policy will prevail, and the additional cost of gasoline production will be recovered by an increase in the price of all transportation fuels and equivalent products. In other words, the traditional price spread between gasoline and diesel fuels will be maintained, so that a price increase in gasoline will create similar increases in diesel, jet fuels and heating oil prices.

On this basis, it is expected that the reduction in the use of lead antiknock compounds will produce the price changes shown in Table IV.VIII.

The forecast price increase for 1993, when lead is to be virtually eliminated, is subject to several variables that will tend to reduce the quoted figure of 2.1 cents per litre (which covers the whole cost of the effects of the new motor vehicle regulations). It is probable that the petroleum industry will develop some advances in technology of processes, additives and catalysts that will reduce the 1993 cost significantly. Such developments are beginning to emerge in the United States, where new regulations on lead came into effect in July, 1985, and January, 1986.

Table IV.VIII

Estimated Increase in Retail Price of Gasoline

Year	Use of lead antiknock compounds	Retail price increase (cents per litre)
1987	Reduction to 0.29 g/L in 1987	0.5
1990	0.29 g/L plus 90/10 population of unleaded/leaded vehicles in 1990	1.0
1993	Virtual elimination of lead in gasoline* by January 1, 1993	2.1

Note: Prices are in current dollars based on 1985 sales-tax regime.
* Includes the whole cost of phase-down from 1986 levels.

Source: DRI, 1985

Notwithstanding the use of alternative additives and blending components to replace lead, the industry will be faced with major expenditures to build new octane-producing facilities. The planning, design and construction of such facilities will take at least 3 years, and this must be recognized in setting the timetable for reducing lead in gasoline. The long-term objective agreed to by industry and government should allow refiners to choose the most cost-effective solution to their gasoline quality problems.

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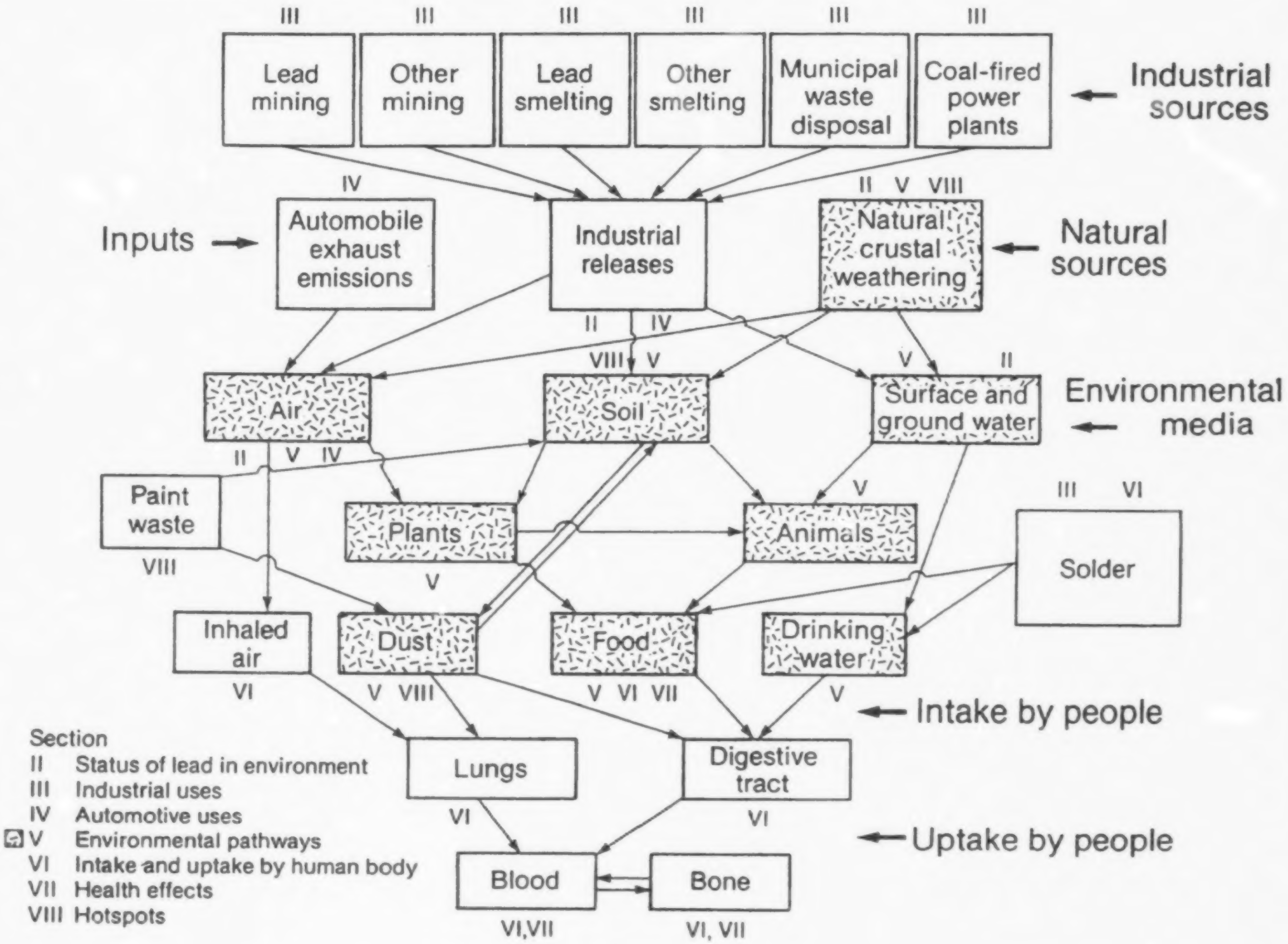
Shading indicates specific
sections (numbered) of text


Industrial sources
III Lead mining
III Other mining
III Lead smelting
III Other smelting
III Municipal waste disposal
III Coal-fired power plants
Inputs
IV Automobile exhaust emissions
Industrial releases
II V VIII Natural crustal weathering
Natural sources
II IV VIII V
Environmental media
Air
II V IV
Soil
Surface and ground water
V II
Paint waste
VIII
Plants
V
Animals
V
III VI Solder
Inhaled air
VI
Dust
V VIII
Food
V VI VII
Drinking water
V
Intake by people
Lungs
VI
Digestive tract
VI
Uptake by people
Blood
VI,VII
Bone
VI, VII
Section
II Status of lead in environment
III Industrial uses
IV Automotive uses
▨ V Environmental pathways
VI Intake and uptake by human body
VII Health effects
VIII Hotspots

Area covered in Section V

V How Lead Reaches People: Environmental Pathways

1. Pathways and Cycling

In nature, lead is chiefly confined to large ore-bodies far removed from human contact, or diffused sparsely through crustal rock and ocean waters. To reach human subjects it must move along identifiable pathways towards its target. Long afterwards it will be returned again to the natural storage reservoirs.

In the course of this passage lead may be considered to move through specific compartments as it progresses from source to ultimate receptor. While in transit, it will be subject to physical and chemical transformations that result from both biotic and abiotic processes. The Commission has reviewed these movements and processes in detail in its Pathways Workshop Report (PWR), "Pathways, Cycling and Transformation of Lead in the Environment"*. Figure I.1 (see Section I) shows the conceptual model within which these ideas fit.

2. Sources of Lead: Anthropogenic versus Natural

The major human-made (anthropogenic) sources of lead were discussed earlier in the report. In addition, natural processes, such as:

- weathering of crustal material
- erosion and loading of soil
- volcanic and other geothermal emissions
- forest fires

may mobilize lead into air, soil and water.

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* Bibliographic references are cited by author and PWR.

Anthropogenic global emissions to the atmosphere have been estimated to be 331 000 t/a, whereas natural sources yield only 24 500 t (Nriagu, PWR). The nature and scale of the natural sources are not well-known, but they are clearly much smaller than the anthropogenic sources. Human beings are the real mobilizers of lead--and also among the most sensitive targets.

Whether natural or anthropogenic, releases of lead into the environment can usefully be categorized according to the medium that first receives them. Table V.I summarizes the information presented thus far in Sections III and IV. It will form the basis of the following discussion.

3. Pathways to People

In the following sections, the major vectors (i.e. transporting routes and agents) from air, water and soil are considered systematically, omitting those pathways going directly to man. Direct pathways are dealt with in Section VI, which discusses uptake.

(a) Lead Delivered via the Air

Processes that release lead into the air, described in detail in Sections III and IV, include mining and smelting, incineration, gasoline burning, battery manufacturing and sand-blasted or flaking paint. In addition, soils may be a source of airborne lead on a local scale, as suggested by the strong correlation between lead in dust (PbD) and lead in soil (PbS)*. The re-entrainment of deposited lead in dust is, however, unlikely to be an important net source to the atmosphere, because large particle size limits dispersal.

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* Not to be confused with lead sulphide.

Table V.1

Summary of Sources of Lead for Air, Soil and Water

Process	Release (t/a)*, physical/chemical form		
	Into air	Into, onto soil	Into water
Natural weathering	**	**	**
Natural combustion (volcanoes, etc.)	**		
Forest fires	**		
Lead mining and milling	964** dust, particulates	$38x10^3$** tailings	49** mine drainage
Other non-ferrous metal mining and milling	1706** dust, particulates	**	**
Iron and steel manufacturing	600** dust, particulates		9**
Lead smelting and processing	304** dust, particulates	$17x10^3$** slags	29**
Other non-ferrous metal smelting and processing	** dust, particulates		
Secondary lead smelting	14**	**	
Municipal sewage treatment/effluent			**
Municipal sewage treatment/sludge	2**	15**	412**
Municipal garbage disposal/land-filling		**	
Municipal garbage disposal/incineration	67**		
Coal-fired power plants	14**		
Alkyl lead production	42**		37**
Alkyl lead use	7173**		
Battery manufacturing	20**		4**
Paint manufacturing	1**)		
Paint consumer use	)	100**	
Solder - manufacturing			
Solder - plumbing use			**
Solder - food and beverage (can) use			
PVC manufacturing			
Cement manufacturing			
Others			

* Chiefly from Section III.
** Process releases directly into the medium as indicated.

Source: This report, chiefly
Sections II and III

Size fractionation of air particles reveals a predominance of lead in the submicrometre fraction, with a remarkably similar size distribution among urban, non-urban, overland and marine sites. Davidson (PWR) pointed out that this suggests a common type of source, i.e. combustion processes. Actual concentrations in air, however, differ markedly among sites, with ranges typically 1-10 $\mu g/m^3$ in urban areas, and 0.1-1.0 $\mu g/m^3$ in rural areas. Figure II.1 (see Section II) shows the time trend of concentrations of lead in air for Canada. Plants are direct recipients of airborne lead in the form of wet and dry deposition. This fact, combined with certain properties of simple (cryptogamic) plants such as mosses and lichens, has been applied in biomonitoring* for lead, as well as for many other elements (Martin and Coughtrey, 1982).

Mosses and lichens, lacking cuticle, and with limited uptake of materials from their substrates (soil, rock or other surfaces), absorb a great deal of material deposited from the atmosphere onto or into their tissues. Hence they provide a means of measuring the deposition of material. Aside from any effects on the plants themselves, or on consumers of the plants, there has been considerable interest in the use of these plants to determine spatial and/or temporal trends in metal concentrations in the atmosphere (Puckett, PWR).

Empirical relationships have been demonstrated between concentrations of lead in plants and distance from source (e.g. Ruhling and Tyler, 1968; Takala and Olkkonen, 1981). Furthermore, identification of source, or of relative contribution from multiple sources, has also been attempted, mainly by the use of enrichment factors (EF's). An EF is determined by comparing the ratio of the concentration of the element to that of a reference element (e.g. titanium, scandium) in the lichen, with the same ratio in crustal rock. This approach has been used extensively to identify natural and anthropogenic sources contributing to the atmospheric burden of elements (Lannefors et al., 1983).

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* Using species of plants or animals as collectors of pollutants, thus providing surrogates for measurements of available concentrations in the environment.

Puckett (PWR) identified three categories of source for spatial monitoring of lead deposition: line sources, point sources and remote sources. Of these, studies on remote sources represent the most effective demonstration of the use of lower plant biomonitors. The author summarized a large number of studies drawn from Finland, Denmark, Norway, West Germany, Holland, eastern Canada and eastern North America. One such study by Steinnes (1977), which included lead among 28 elements, showed a clear south-to-north gradient of lead in the moss Hylocomium splendens in Norway. With no major sources of lead in southern Norway, the data were interpreted as showing that sources outside the country were providing the lead and that the atmosphere was the pathway. Other elements showed a similar pattern. Studies from elsewhere show similar, though sometimes less clear-cut, patterns, because local or internal industrial sources of lead in air are present in most countries or broad geographic regions.

A major application of plant biomonitors might be to determine temporal changes resulting from controls on emission sources of lead, both locally and long-range. In fact, temporal studies on Sphagnum (Ruhling and Skarby, 1979) have already suggested decreases in lead in these mosses over the period 1968-1975. Steinnes (1986) recently communicated to the Commission that preliminary results for the 1985 survey in Norway indicated that lead levels in _H. splendens_ had decreased by a factor of two from his 1977 survey.

Measurements of the chemical and physical forms of lead in the atmosphere have been made in only a few instances, apart from the particle size fractionations described by Davidson (PWR). Even for these, the author expressed some reservation about the acceptance of early data. The separation of particulate and volatile forms of lead in air has potential problems, because the normal method of collection from air, i.e. filters, will not catch all the volatile fraction. Alkyl leads in air are mainly in volatile forms, and most inorganic lead is particulate, though some of the alkyl lead in the atmosphere may be included in the particulate form (Van Loon, PWR).

Techniques for determination of different chemical forms (species) of lead in air are being developed in several analytical chemical laboratories (e.g. Radziuk et al., 1979). It is to be expected that the different chemical forms of lead in air will behave differently as regards deposition and biological availability, though coagulation of particles in the atmosphere probably decreases the variability of chemical forms. There are at present no published data relating the chemical forms of lead in air to their respective behaviours in the environment. It is known from experiments that tetraethyl lead (TEL) is more toxic than are inorganic forms (Hodson, PWR), but integrated environmental data on the relationship between chemical and biological data on lead in air are lacking.

Lead deposited from the air and subsequently incorporated into plant tissues may enter the food web and find its way into wildlife or domestic animals, which eat the plants. The same is true for people consuming wild and cultivated fruits and vegetables. There are few studies of the effects of lead in air on terrestrial herbivorous wild animals and birds. Considering the technical problems of separating different sources of exposure for non-experimental animals, this is scarcely surprising.

Southwood (1983) tabulated several studies of lead in animals grazing beside roads, where elevated lead concentrations in various tissues were related to distance from the road, but where direct uptake of airborne lead as well as lead from vegetation could not be factored out. Ward et al. (1978a, b) demonstrated elevated blood lead levels in sheep grazing beside roads in New Zealand, and concluded that both pathways were operating. However, the air-vegetation-animal pathway is likely to be more important for herbivores than it is for man, because of the high surface/volume ratio of grasses compared with human food crops. Hence, for roadside animals the food vector is the most important one. Steinnes (PWR) showed that lead in the livers of lambs collected in different parts of Norway reflected the deposition of atmospheric lead; again, however, it was not possible to resolve the relative importance of the two possible pathways.

Lead in the liver of wild moose, reindeer and red deer was studied by Froslie et al. (1984) in Norway. The south-to-north gradient of lead described already for mosses in Norway was reflected in moose liver, but not in livers of other animals, i.e. the lead source for the moose was apparently atmospheric, but the pathway could still have been air and/or food. According to these workers, the mainly western distribution of the red deer, and the feeding habits of the reindeer, apparently overwhelmed the effects of atmospheric lead for these other herbivores. As far as actual concentrations of lead in tissue were concerned, reindeer had the highest concentrations, and the authors ascribed this to feeding habits. Reindeer feed on grasses in summer and lichens in winter, and lichen pastures were suggested as the major source of lead for these animals. Lichens not only have high surface/volume ratios, but also grow very slowly, so they may accumulate lead as well as other metals for a long period before they are grazed. This seems to be the only example where evidence, albeit indirect, is presented for food-chain accumulation of atmospheric lead in wild animals.

There is a consensus that lead is not biomagnified (increased in concentration with increasing trophic level*) in the food chain; in fact, the reverse is probably true. This will be discussed in more detail in the section on aquatic food-chains (p. 136). There appear to be no instances of lead poisoning in wild animals resulting from airborne lead, with the exception of highly contaminated situations such as smelter sites. For the most part, lead from the atmosphere accumulates in soil and persists there. It can inhibit invertebrate and microbial activity. This is discussed in the section on soils (p. 125). Southwood (1983) reviewed instances of lead poisoning in farm and domestic animals, but none of these was related to airborne lead as the immediate source. Soils, paint and old mining waste were the apparent sources. One such case is known among horses at Trail (Cominco Ltd., 1985).

Fruits and vegetables, like lower plants, can absorb lead from the atmosphere. The concentrations in these food crops are likely to be lower than in mosses and lichens (Table V.II), for several reasons. Firstly, the waterproof cuticle on the surface of higher plants limits absorption of metals, especially in wet deposition; secondly, most food crops have a fairly short time span from planting to harvest; and thirdly, most plant foods are

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* i.e. from the plants upwards to herbivores, and on to carnivores.

Table V.II

Lead Concentrations in Plants, Excluding Experimental Situations and Smelter or Roadside Sites

Plant	Range (µg/g dry weight)	Reference
Lichens	5-65	Steinnes, PWR
Mosses	11-62	Gydesen et al., 1983
Leaves of perennial woody plants	0.3-9.6	Steinnes, PWR
Roadside grass (excluding highly contaminated sites)	5-90	Ratcliffe and Beeby, 1980
Cabbage (leaves)	<0.01-0.51	Southwood, 1983
Collards (leaves)	2.6-6.6	Chaney et al., PWR
Tomato (fruit)	0.01-0.14	Southwood, 1983

washed or peeled in preparation for the table. Nevertheless, air as a potential source of lead in food, affecting growing crops as well as fruit and vegetables displayed in markets and stores, represents an important pathway towards people.

The significance of airborne contaminants for grazing animals has been brought into sharp focus recently, as a result of the deposition of ^{137}Cs from the Chernobyl accident in the Ukraine. In Norway (Steinnes, 1986), sheep and reindeer are contaminated with the radioactive material such that their human consumption is no longer safe. These animals, grazing on grass and lichens, respectively, have accumulated the contaminant as a result of long-range atmospheric transport followed by accumulation in the plant tissues that are their food base. The situation in Finland is equally serious. In these instances, the effect of the contaminant on the animals themselves is likely to be much more severe than for lead because of the potential for radiation damage. The relevance of this incident to the lead issue, however, is in the area of bioaccumulation and food-web transport to man of airborne contaminants in general.

Much discussion has focussed on the relative importance for plants of directly deposited atmospheric lead versus the route through the soil. Southwood (1983) concluded that "it is at present not possible to quantify the relative contributions made by the various routes" (from soil and through deposition on aerial surfaces, the latter from both the air and by soil splash). However, in order to quantify the significance of the various sources and pathways to man, some attempt will be made to estimate the relative importance of the routes.

Chaney et al. (PWR) pointed out that lead is one of several elements not readily available to plants in most soils. Furthermore, lead is not readily translocated through plants. Ter Haar (1970) showed that filtering the air did not change the lead content of most of the vegetables that he tested, though air lead was a source for non-edible parts of certain plants. Studies attempting to separate air and soil as sources of lead for plants are often confounded or obscured by other factors, e.g. by differences in other soil factors such as fertility and pH. The most recent studies reported by Chaney et al. (PWR) nevertheless concluded that, for leafy vegetables, "aerosol deposition usually exceeds soil as the source for lettuce, collards and other leafy vegetables growing in urban gardens." However, splash from soil has also been considered as a potential source of lead for leafy vegetables.

The residence time of lead in the atmosphere is short (about 10 d in the Northern Hemisphere), and so atmospheric concentrations are not good indicators of environmental lead burden. Nriagu (PWR) pointed out that "the rate of lead fallout should be regarded as a better measure of the environmental lead burden" than the concentration in air. Recently, a lead fallout standard of 250 $\mu g/m^2/d$ has been proposed in Germany (Chamberlain, 1986). Lead falling from the atmosphere, if it is not inhaled, and if it does not enter the food-chain from deposition onto plants, has to be traced through soils and surface water systems in which it accumulates.

(b) Lead Delivered via the Soil

Sources of lead in soils include the atmospheric sources described earlier and, in addition, natural geological sources, mine wastes, deliberate application of sewage sludge as a fertilizer, application of commercial fertilizers, peeling, chipping or finely ground (sanded) paint and surface and subsurface disposal of a variety of domestic and industrial wastes.

The range of concentrations of lead in soils is immense, even excluding remote areas. Values range from less than 5 µg/g in rural areas to several per cent close to smelters. Table V.III shows selected data. Caution is required in comparing absolute values among studies; though analytical limitations are rarely a problem, sampling can influence the result in a profound way. This is due in part to the fact that lead is rather immobile in soil, and additions from airborne sources tend to show a profile effect with high concentrations near the surface (Table V.IV). A surface grab from the top 5-10 cm will hence give very different values from a core of 20 cm or a surface scraping. Furthermore, because lead tends to accumulate in the organic humus layer, inclusion or exclusion of this layer in or from a soil sample will have a major effect on the reported concentration.

Management of soil will, of course, have another effect on the measured values. Tillage or cultivation, which mixes soils to a depth of 10-20 cm, will effectively dilute the lead entering from the atmosphere or applied in sludge, so that agricultural soils may be, other things being equal, safer than undisturbed soils.

Problems created by possible solutions for lead-contaminated soils are discussed in Section VIII. At this stage, it should be noted that the risk to people through the soils pathway is strongly influenced by many factors, including land use, soil type, personal habits, age and socio-economic status. These will be referred to in Section VI on direct pathways to man, as well as in the context of source/blood lead relationships.

Table V.III

Lead Concentrations in Soils: Selected Values

Type of area	Lead concentration (μg/g)	Reference
Canada (remote)	5-20	Nriagu, PWR
Canada (rural)	10-50	Nriagu, PWR
Canada (inner-city)	150-3000	Nriagu, PWR
Canada (around smelter)	3%	Nriagu, PWR
Ontario (rural, 0-5 cm)	5-360	Rinne, 1986
Ontario (urban, 0-5 cm)	5-845	Rinne, 1986
New Brunswick (podzol L-H horizon)	0-100	Presant and Tupper, 1965
New Brunswick (podzol above sulphide deposit L-H horizon (mean))	1041	Presant and Tupper, 1965
UK (uncontaminated)	10-150	Southwood, 1983
UK (urban, London)	671	Thornton, 1983
UK Shipham (old 18th-century mining area)	3800	Southwood, 1983
US Missouri lead belt, close to smelter	30 400 (max)	Wixson, 1977
Ontario, close to secondary lead smelter	21 200	Linzon et al., 1976

Table V.IV

Lead Concentrations in Some Soil Profiles
(all in µg/g dry weight)

I. Natural sources, New Brunswick, Canada*

Horizon	Above sulphide deposit	Not above sulphide deposit
L-H	1041	73
Ae	752	13
B_1	1290	36
B_2	1221	28
C	1431	65

II. Contaminated soils, Kingston, Ontario (smelter closed 1916)**

Profile	Various sampling sites		
Surface	8516	594	11666
1-5 cm	1863	455	3208
5-10 cm	1156	261	167

Sources: * Presant and Tupper, 1965

** Stokes, 1977

Invertebrates and microflora are undoubtedly affected by lead in soil. Evidence for this comes mainly from studies on smelters, and is therefore circumstantial, because lead is rarely the only contaminant. In a study of Crooked Creek Watershed in Missouri, Watson et al. (1976) demonstrated a distance-related effect of the primary lead smelters and the lead mine in the New Lead Belt on soil arthropod populations. Recent studies in the Adirondacks (Siccama et al., 1980) have implicated increased lead in decreased rates of litter decomposition, though a cause-effect relationship was not established. Toxicity of lead to plants has been described at soil concentrations ranging from 35 µg/g ("reduced growth"; Davies, 1978) to 500 µg/g ("toxicity to trees"; Jaworski, 1978). Most of the data on toxicity to plants come from plants exposed to lead in soil in laboratory or greenhouse experiments, and plant cover is frequently found in soils containing several thousand parts per million of lead. There is great variation among species, or even within species, in response to lead. The absence or loss of plant species from polluted soil is only circumstantial evidence for toxic effects of lead, as other factors such as soil pH, soil contamination by other metals and air pollution, e.g. by ozone or sulphur dioxide, usually accompany increased levels of lead.

In spite of this, fairly clear indications in lead-rich soils of lead toxicity, or selection of certain ecotypes, are available for smelter and roadside studies (Table V.V). Such occurrences are quite rare, or at least represent very specialized situations. Though of fundamental scientific interest, they are less important in the present study than the uptake and accumulation by normal plants. The uptake by plants of lead from soil usually occurs without any toxic symptoms, and is thus a more insidious type of risk,

Table V.V

Examples of the Evolution of Resistance or Tolerance to Lead

Species	Effect	Reference
Marchantia polymorpha (liverwort)	Tolerance to lead varies with lead in the soil	Briggs, 1972
Plantago lanceolata (dicotyledonous weed)	Lead tolerance in ecotypes from roadside soil	Wu and Antonovics, 1976
Agrostis tenuis (bent grass)	Lead resistance in ecotypes from lead-zinc mine spoil	Bradshaw, 1952
Anthoxanthum odoratum (vernal grass)	Lead tolerant clones from lead-zinc mine spoil	Cox, 1976
Asellus merideanus (aquatic isopod)	Lead tolerant variety from a river contaminated by old lead-zinc mine drainage	Brown, 1977

especially to consumers of the plants.

Most research into such uptake, particularly in the last decade, has focussed on vegetable crops. Earlier work in the 1970's looked at metals in soil from sewage sludge. A considerable body of information in this area is reviewed in detail by Chaney et al. (PWR). Sewage sludge is applied to agricultural land as a fertilizer and as a source of organic matter to condition soil. The organic component of the sludge binds metals, so that any lead entering the sewage treatment plant is concentrated in the sludge. In fact, adding sludge to soil, even when there is a net addition of lead, may decrease lead uptake because of the additional organic matter capable of binding lead. The source of lead in sewers includes industrial waste and run-off from roads into storm sewers. Concentrations of lead in sludge ranging from 13 to 26 000 µg/g have been recorded (Logan and Chaney, 1984), but most political jurisdictions in developed countries have recognized the potential risks, and metals in sludge applied to agricultural land are regulated. The objective is to prevent phytotoxicity and transfer of metals to man via vegetables and fruits.

Plant accumulation of lead from sludge-amended soils, providing good practices are used, appears to be minimal, because plant lead concentrations in such situations are comparable with those of plants growing in unamended soil. Concentrations of lead in lettuce grown on sludge-amended soils with concentrations of lead in sludge ranging from 215 to 865 µg/g never exceeded 3.3 µg/g, and there was no relationship between soil lead and plant lead concentrations (Chaney et al., PWR). The high organic content of sludge and usually neutral pH of agricultural soils in Canada both tend to reduce lead uptake into plants from agricultural soils receiving sludge. Sludge guidelines are reviewed in Section VIII.

Concern has recently arisen over lead in garden crops. Renewed interest in producing home-grown vegetables, along with increased research into lead toxicity, has stimulated a reconsideration of the soils-to-people pathway in urban and industrial areas. There is, in fact, little evidence that garden vegetables contain high concentrations of lead, even for inner-city or highly polluted soils (Table V.VI), except possibly in outer leaves and roots, which are not normally eaten. Contamination may result in part from soil splash onto leaves rather than from root uptake from soil. There is much more cause for concern about children who suck dirty fingers, or even eat soil (pica) (see Section VIII). Nevertheless, lead from anthropogenic sources has accumulated, and in some areas continues to accumulate, in soils used to raise crops. Some quantitative estimates of this pathway have been made.

Pathways from air to vegetables are estimated through the following transformations:

- Air as a source for soil. Soil accumulation depends upon:
    - ° deposition rates of particles (varies with size)
    - ° concentration of lead in air
    - ° rate of addition of material to soil (burying)
    - ° leaching from soil
    - ° plant uptake from soil
    - ° addition of plant material to soil (recycling of lead)

  A model developed by EPA (1986) relates soil lead levels closely to air lead concentrations, as shown in Table V.VII. In the ESSA model developed for this Commission, a similar dependence is assumed.

Table V.VI

Concentrations of Lead in Crop Plants in Relation to Lead in the Medium

Plant species	Type of study	Concentrations of Lead (µg/g)		Reference
		In medium	In plant	
Bromegrass	Field-collected smelter-contaminated soils	680	4.9-34.5	Marten and Hammond, 1966
	Soil from near highway	59	2.7-6.5	
	Control soil	12	3.0-5.4	
Lettuce (tops)	Freeway soil and traffic	97	20	Rabinowitz 1974
	" " " "	280	30	
	" " " "	1300	80	
Oats (tops)	" " " "	97	125	
	" " " "	280	125	
	" " " "	1300	300	
Bean (top)	Experiment + solution added lead	1	3.52	Broyer et al., 1972
(root)	" "	1	225.74	
Bean (top)	" "	10.0	72.28	
(root)	" "	10.0	1103.84	
Barley (top)	" "	1.0	4.35	
(root)	" "	1.0	258.88	
Barley (top)	" "	10.0	38.43	
(root)	" "	10.0	2588.75	
Tomato (top)	" "	1.0	1.45	
(root)	" "	1.0	232.99	
Tomato (top)	" "	10.0	6.21	
(root)	" "	10.0	1467.45	

Table V.VII

Relationship Betwee.. Soil Lead and Air Lead Concentrations

	Concentrations of lead (µg/g)	
	Air	Soil/dust
General	0.25	55-200
Point source	0.25	100-525
General	0.5	150-500
Point source	0.5	350-800
General	0.75	275-875
Point source	0.75	600-1125
General	1.0	500-1150
Point source	1.0	750-1450

Source: EPA, 1986

- Uptake of lead from soil by plants through roots. Uptake by this pathway is slight. Attempts to quantify it have been made by use of a concentration factor (CF) (Chamberlain, 1983):

$$CF = \frac{\text{concentration of lead in plant}}{\text{concentration of lead in soil}}$$

Values for CF range from less than 0.002 to greater than 0.2, but rarely exceed 1.0. CF's decrease with increased soil lead, and are also affected by physical and chemical factors. Measured CF's do not, however, give reliable indication of the soil-to-plant-root uptake, because addition of lead from soil may occur by direct contamination of the above-ground parts, especially by the splashing of soil lead onto leaves and stems by rain.

- Foliar uptake of lead. Though aerial deposition of lead onto the plant surface, usually the leaf, does certainly result in lead uptake, there is uncertainty about the relative importance of soil and foliar routes. Chamberlain (1983) proposed the use of the accumulation rate (AR):

$$AR = \frac{\text{concentration of lead in foliage } (\mu g/g)}{\text{concentration of lead in air } (\mu g/m^3)}$$

In urban areas, values of AR of 20 have been given where deposition rates reach 900 $\mu g/m^2/d$ (Fergusson, 1986). For rural areas, by extrapolation, AR's would be in the order of 6 (Chamberlain, 1983).

(c) Lead Delivered via Water

Natural sources of lead in water are quite weak compared with anthropogenic sources. Lead may be discharged directly into water, either as routine practice or accidentally, from a number of industrial activities (see Sections III and IV and Table V.I). Data from Canada are included in studies of the release of TEL near Maitland, Ontario (Hodson, PWR) and the release of inorganic lead from mines in Nova Scotia (Westlake et al., 1982) and New Brunswick (Ray, 1978). Each of these activities resulted in elevated lead levels in water, which apparently affected fish. However, in the last two examples, elevations of zinc and copper also occurred, making interpretation of the effects of the lead on fish quite difficult. For field studies it is important to develop some specific biological indicator (e.g. blood lead levels in fish) to provide hard evidence for relationships between a particular toxicant and effects, or potential effects.

Of much greater significance than point-source discharges of lead for aquatic systems is the deposition of lead from the atmosphere, and the transport of lead overland from soils and other surfaces. For example, Leland et al. (1974) estimated that more than 90% of the lead input to southern Lake Michigan could be attributed to aerosol lead deposited in precipitation and run-off. More recently, Hodson (PWR) stressed the significance of the atmosphere rather than mining activities as a source of lead for Lake Ontario.

Concentrations of lead in surface waters are generally low, in the order of 1-12 µg/L in the Great Lakes (Hodson, PWR; Jaworski, 1978). They are usually lower in marine than in fresh waters. The total global burden of lead in fresh waters is 1.2×10^6 kg, and in the oceans 1.4×10^9 kg (Nriagu, PWR). Of interest in terms of the source of lead is its vertical distribution in the open ocean, which Nriagu (PWR) described as being higher in surface layers than in deep waters. Furthermore, lead concentrations in the surface waters of the North Atlantic are 10 times greater than those in South Pacific surface waters (Settle and Patterson, 1982; Boyle and Huested, 1983; Bruland and Franks, 1983).

Direct deposition from air and precipitation and influx of suspended sediment from rivers combine to contribute to a net increase in the lead burden of aquatic systems over time. Estimated influx to oceans from all sources is 227×10^6 kg/a, whereas losses are only 20×10^3 kg/a (Nriagu, PWR).

Palaeoecological studies of lead in lake sediments indicate that the first major increase in deposition rate in the North American continent began about 100 years ago, with a further increase in the early 1940's. There has been a slight decline over the past 15 years (Norton, PWR). For Great Britain, research based upon similar techniques has shown that the first increase occurred much earlier, reflecting the earlier industrialization in that country. Both fossil-fuel combustion and metal smelting contributed to the sediment lead burdens in the first rise in deposition rates. The introduction of TEL resulted in the most recent rise. The decline observed over the past decade or so cannot be attributed solely to controls on TEL, but tend to reflect a general improvement in air lead concentrations, traceable to many causes (see Figure II.1, Section II).

One of the reasons for the generally low concentration of lead in water is the tendency for the metal to become partitioned into the solid phase by precipitation or adsorption onto particulate material. For example, Kinniburgh and Jackson (1981) showed that lead adsorbed more strongly to $Fe(OH)_3$

gel than did copper, zinc, nickel, cadmium or cobalt, and that lead required a lower pH than any of these other metals for half the adsorbed metal to be released.

The concentration of lead in bed sediments of streams and lakes ranges from less than 1 to 950 μg/g (dry weight), excluding highly contaminated sites (Jaworski, 1978), and sediments have been regarded as sinks for lead as well as for other metals. Though the quality of overlying water certainly benefits from the tendency of lead to become deposited into sediments, there are many unanswered questions about the recycling of lead from sediment, especially via sediment-dwelling biota, and about the actual fluxes of lead out of sediments of rivers, lakes and ultimately oceans. Biological and chemical processes that respectively stabilize or mobilize lead, and methods for determining the chemical forms of lead in sediment, are addressed later in this discussion.

Lead in water _per se_, however, even if relatively low in concentration, can represent a risk to people and biota. Water containing dissolved lead has been identified as a dietary source of lead for people, either through consumption of drinking water or other beverages, or from eating vegetables cooked in lead-contaminated water. In most quantitative dietary lead budgets, water is represented as rather insignificant (e.g. EPA, 1986). The major excursions from background levels result from lead _added_ to water in the course of water distribution or collection (see Sections II, III and VIII).

Another potential source of lead for people via the water pathway is the consumption of fish and shellfish. Seaweeds may also make a contribution to dietary lead in certain geographic areas and cultural groups. The toxic effects of lead on biota may also affect aquatic ecosystems and wildlife, and may have socio-economic effects. Data on most of these impacts are not difficult to find, but useful dose-response data are much less common.

The ranges of lead concentrations that are toxic to algae, invertebrates and fish are extremely large, even within a given group of organisms. They have been summarized recently by Hodson (PWR) (Table V.VIII). One reason for these differences may be variation in the chemistry of lead in water, but there are also real differences among species of organisms in their response to lead.

The chemistry of lead in natural waters was reviewed by Jaworski (1978), who discussed theoretical and analytical aspects of lead speciation. The tendency for lead to form complexes with organic as well as inorganic ligands, and the pH and pE (oxidation potential) dependency of solubility and speciation of lead compounds, mean that a number of studies of lead toxicity in natural waters or in unspecified media are difficult to interpret. Furthermore, the alkyl leads are much more toxic and bioavailable than are inorganic lead compounds (Hodson, PWR). Alkylation processes for Pb(II) in the environment remain quite controversial, though the methylation of Pb(IV) is apparently accepted (Chau, PWR).

In general, recent attempts to integrate end-points for toxicity tests, such as the biomass production rates of Borgmann et al. (1978), indicate limits of 48 μg/L for snails and 100 μg/L for zooplankton, i.e. levels exceeding the concentrations observed in most surface waters. However, Hodson (PWR) showed behavioural effects on fish at much lower lead levels, i.e. 3.8-10 μg/L, which are not uncommonly encountered in industrial or heavily urbanized situations. At Maitland, Ontario, where TEL was released into the St. Lawrence River, the blood lead level of fish in the affected reaches was as

Table V.VIII

Examples of the Toxicity of Lead to Aquatic Organisms

Organism	Concentration resulting in toxic effect (μg/L)
Algae	10 - 100 000
Invertebrates	28 - 71 000
Fish (LC_{50})*	200 - 542 000

* Concentration lethal to 50% of the test population.

Source: Hodson, PWR

high as 649 μg/L, compared with 48 and 64 μg/L for control areas (Hodson, PWR). It does not appear at present that fish and other aquatic biota are at much risk from lead toxicity, but more information is needed, especially concerning point-source emissions and alkyl lead.

Concentrations of lead in tissues such as blood, liver or kidney often reflect exposure, and can sometimes be related to toxicity; however, this aspect of lead toxicology for biota is still at an early stage of development. What is consistently clear, however, is that lead does not accumulate in the edible (muscle) portion of fish. This is obviously very important for the quality of fish as human food. Another fact also consistently shown in studies of lead in aquatic systems is that lead is not biomagnified. The concentration of lead in primary producers is generally higher than that in primary consumers, and top predators generally contain the least lead if it is expressed on a concentration per unit weight basis. In fact, the term "biominification" (systematic decrease in concentration with increasing trophic level) has been applied to the behaviour of lead in food webs.

The source of lead in water for consumers includes not only water, but also food items, which may be other biota, detritus or inorganic particles. Bottom-feeding (benthic) fish commonly have higher body burdens of lead than do pelagic feeders from the same aquatic system, as shown, for example, by Hodson et al. (1983) and Czanneski (1985). Benthic invertebrates are the organisms having the greatest potential for exposure to lead, because their milieu generally contains the highest concentrations of lead, and they feed in or on the solid phase of the sediment.

Exposure to lead in sediment could result in toxic effects, either possible loss of species (i.e. changes in ecosystem structure) or bioconcentration (uptake) of lead, or both. The exposure of benthos to lead is of interest for three major reasons. Firstly, marine and occasionally freshwater molluscs and crustaceans are used as food for man. Secondly, the benthos play a crucial role in the cycling of carbon and other major nutrient elements in aquatic systems. Thirdly, on a practical level, the benthos can be used as biological monitors for lead in aquatic systems.

Only a small part of the total lead in water and sediments enters food webs. Factors tending to decrease lead uptake from sediment include high concentrations of organic carbon and iron oxides. For example, in a study of the deposit-feeding bivalve Scrobicularia plana from four estuaries in the UK, Luoma and Bryan (1978) showed that lead in the animal was predicted best by the sediment lead/iron ratio, rather than by total lead or any other operationally defined fraction of lead in sediment.

Unfortunately, it is not possible to predict reliably the amount of lead bioaccumulated by benthic animals. As shown in Table V.IX, there are quite large differences within and between species in terms of lead bioconcentration. Even within the so-called feeding guilds (groups of organisms with similar feeding habits), it can be seen, e.g. for the filter-feeders in Table V.IX, that maximum accumulation by oysters is far less than that for mussels. Similarly, for the deposit-feeders in the same table, a comparison of two species of clam shows that Macoma balthica never contained more than 60 µg/g, whereas the ecologically similar S. plana contained lead at up to several thousand micrograms per gram. Our lack of understanding of the mechanisms of metal uptake underlies our inability to predict concentrations. In general, however, food items from aquatic systems suspected of having contaminated sediment should be checked. Levels in shellfish do in general exceed those in

Table V.IX

Concentrations of Lead in Sediment and Species of Different Feeding Guilds from Three Estuaries

Sample	Lead concentration (µg/g dry weight)		
	Severn estuary	West Looe estuary	Isle of Man
Sediment	88	104	3200
Deposit-feeders			
Scrobicularia plana		189	3000
Macoma balthica	9	61	
Nereis diversicolor	3	25	
Filter-feeders			
Mytilus edulis		45	198
Cerastroderma edule		5	371
Mya arenaria			2222
Grazers			
Littorina littorea	4	17	
Patella vulgata	10	30	
Seaweed			
Fucus vesiculosus	3	17	

Source: Luoma, PWR

fish muscle (Table V.X), and concentrations of lead in some shellfish from marine (estuarine) sites can exceed the level acceptable for human consumption.

Almost nothing is known of the effect of lead on the structure and function of aquatic ecosystems. Most studies provide information on body burdens of lead (usually related to point sources or general urban complexes) in riverine, lake or estuarine food webs, and these are certainly related to lead levels in water and sediment; however, field studies can rarely attribute changes in structure of food webs or losses of species to lead _per se_. The studies that exist usually refer to highly contaminated regions, such as those affected by mining and smelting. In common with similar studies on terrestrial ecosystems, they suffer from confounding effects related to the presence of other metals, and frequently to other major physical or chemical changes that accompany contamination.

In brief, there is little evidence outside severely contaminated areas for biological effects of lead on pelagic or benthic biota. Nevertheless, the fact remains that the burden of lead in sediments of fresh and salt waters, especially estuaries, is very great in certain sites, and strategies are therefore required for evaluation of the potential risk of this contamination. The use of biosentinels, or biomonitors, i.e. organisms that, by accumulating an element, give some measure of availability and dose, is recommended for this purpose. A well-known series of studies, the so-called _Mussel Watch_ (Farrington et al., 1983) has demonstrated how standardized collection and analysis of the marine mussel _Mytilus edulis_ can provide information on the relative availability of lead, as well as many other contaminants, at different sites. At the same time, by use of an organism which is also consumed by

Table V.X

Lead in Fish and Shellfish (not canned) that may be Consumed by People

Organism/Location	Lead level (μg/g)	Reference	Comment
Crab, Wales	0.2-0.3 (fresh)	MAFF, 1982)	Whole organism or claw
Crab, Scotland	0.1-0.74 "	)	
Mussel, Wales	0.2-35.0 "		Soft tissue
Mussel, Scotland	0.2-6.4 "		" "
Plaice, UK	0.13-1.16 "		
Whitebait, UK	0.32-0.52 "		
American oyster	7.1 (dry)	Jaworski, 1978	
Oyster, US east coast	4.7 "		
Oyster, US west coast	7.0 "		
Mussel, US	8.4 "		Much higher levels achieved by experimental addition of Pb
Brook trout	0.37-0.64		
Brook trout	1.1-5.8		Contaminated from snow-mobile exhaust
Carp, St. Lawrence R., Maitland	0.21-57.0	Hodson et al., 1984	Gutted carcass
Carp, St. Lawrence R., Johnstown	0.53-3.4		" "
White suckers, St. Lawrence R., Maitland	0.08-3.8		" "
White suckers, St. Lawrence R., Johnstown	0.20-1.5		" "
Northern pike, St. Lawrence R., Maitland	0.13-0.57		" "
Northern pike, St. Lawrence R., Johnstown	geom. mean = 0.01		" "
Carp, Lake Huron	0.04-0.08		Muscle
Carp, St. Clair R.	0.11-2.19		"

Note: Former Canadian standard for human consumption, 10 μg/g fresh weight in fish and shellfish (discontinued, and not replaced); current standard for human consumption, 3 μg/g fresh weight in fish (WHO, 1972).

people, a check can be made as an early-warning system for excessive lead in a food item. No such widespread application of biomonitors for lead has been developed for freshwater systems, though suitable organisms exist, and individual researchers have carried out studies, e.g. on the Great Lakes (Jackson, 1985).

4. Has Acidic Deposition the Potential for Human Health Effects via Lead?

The environmental movements of lead are related to acidic deposition in two ways. Firstly, net addition of lead may result from long-range transport of the metal in contaminated air masses, followed by deposition in rain, snow and fog, or as dry particles and gases. This deposition may affect receiving surfaces such as roof-tops, paved areas, highways, vegetation, soils or surface waters. Human activities account for a high proportion of the lead that is mobilized into air. Though much of it falls close to the source, nevertheless there is transport through the atmospheric pathway (Atmosphere-Biosphere, 1981). Isolines of lead concentration in rainfall (Figure V.1) display similar patterns to those for hydrogen ion concentration (Figure V.2), showing that it is not simply a local problem, but is due to long-range atmospheric transport.

Secondly, mobilization of existing lead may occur as a result of acidification of soils, vegetation and surface waters, indeed of any receiving surface. Such acidification can result from acidic deposition where natural buffering capacity is low. The solubility and chemical speciation of lead are both strongly pH-dependent (Campbell and Stokes, 1985). The lower the pH (i.e. the more acidic the water), the greater the expected biological availability of the lead. At low pH, the total amount of dissolved lead in-

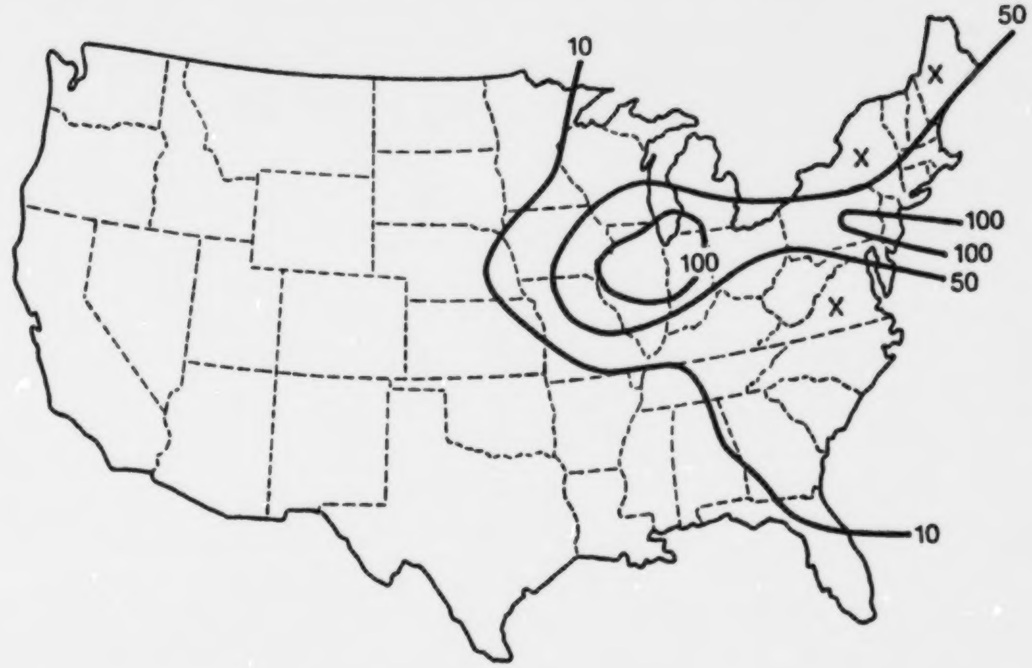
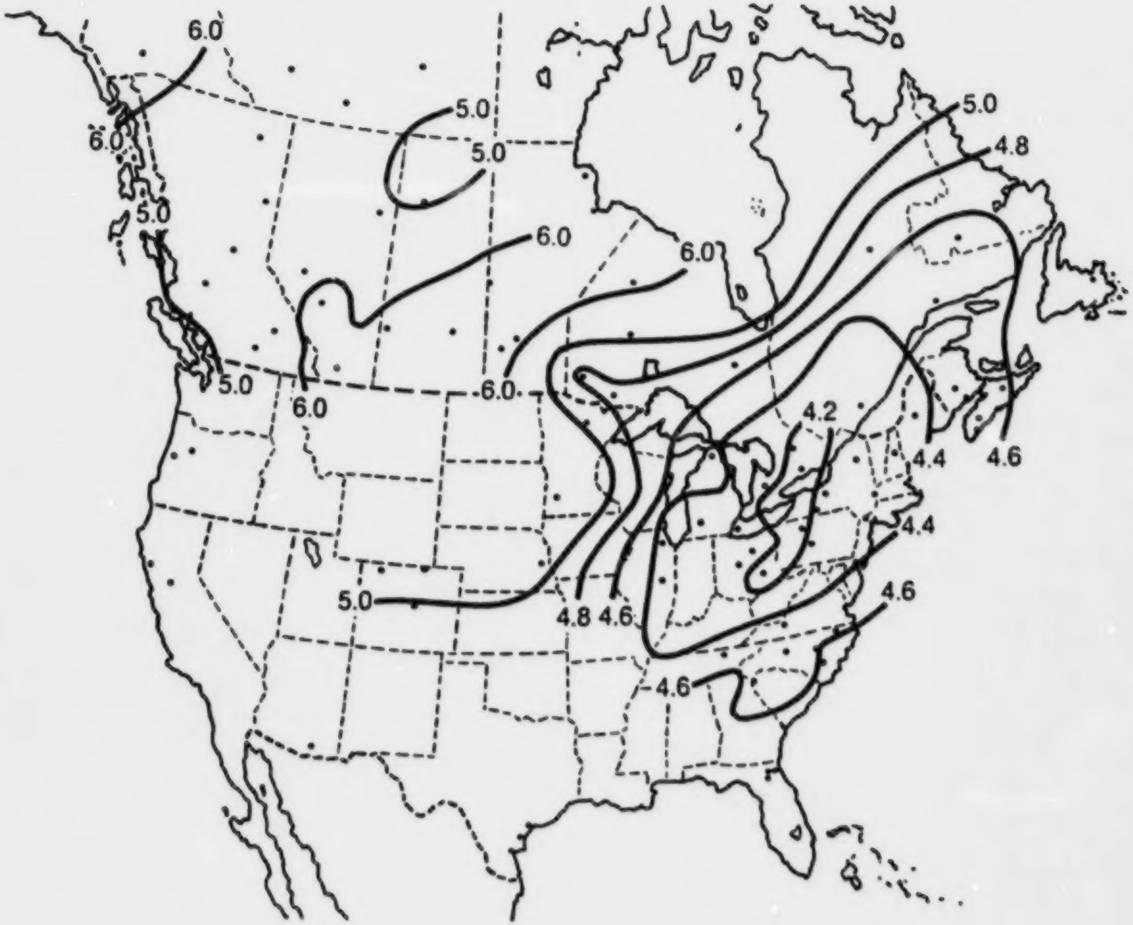

10
50
X
X
100
100
100
50
X
10

Figure V.1. Average lead deposition by precipitation over the continental United States. September 1965 to March 1967 (in grams per hectare per month).

Source: Atmosphere-Biosphere, 1981.


6.0
6.0
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5.0
5.0
5.0
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6.0
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6.0
6.0
5.0
4.8
4.2
4.4
4.6
4.4
4.6
4.6
5.0
4.8
4.6

Figure V.2. Mean annual pH of precipitation in the United States and Canada, 1979-1980.

Source: Atmosphere-Biosphere, 1981.

creases, but this is not apparent until the pH falls below 4.0 (Davis et al., 1982). The source of this mobilized lead may be the deposition processes described in the preceding paragraph, or it may be naturally occurring lead such as that in soils and rocks.

The simplest manifestation of either of these processes is seen in the pH dependence of total lead in surface waters (e.g. Dixon, 1980) (Figure V.3). Actual effects on ecosystems or health have not been unequivocally demonstrated, but can be predicted from models of the chemical behaviour of lead and its toxicity and tendency to bioaccumulate. The present discussion is confined to direct and indirect effects on human health in the context of acidification.

(a) Lead in Drinking Water

The median lead concentration in municipal drinking water in the US is 10 μg/L, but drinking water may contain more than 50 μg/L in certain areas (EPA, 1980). This is related to the presence of very soft, low pH water combined with lead or lead-soldered plumbing in the domestic water system, e.g. in metropolitan Boston (NFEAC, 1982). Flushing out the dissolved lead and other metals can be achieved by allowing the water to run from the tap for a few minutes after a period of standing.

For Canada, recent national surveys of drinking water showed median lead levels of less than 1 μg/L, with a recent range from less than 1 to 7 μg/L (Méranger et al., 1979, 1981; Quinn, 1986). The US and Canadian drinking water standard is 50 μg/L. In areas where cisterns and pipes are constructed from lead, tap water may contain several hundred or occasionally even exceed 1000 μg/L lead (Beattie et al., 1972). Leaching of lead from plastic pipes

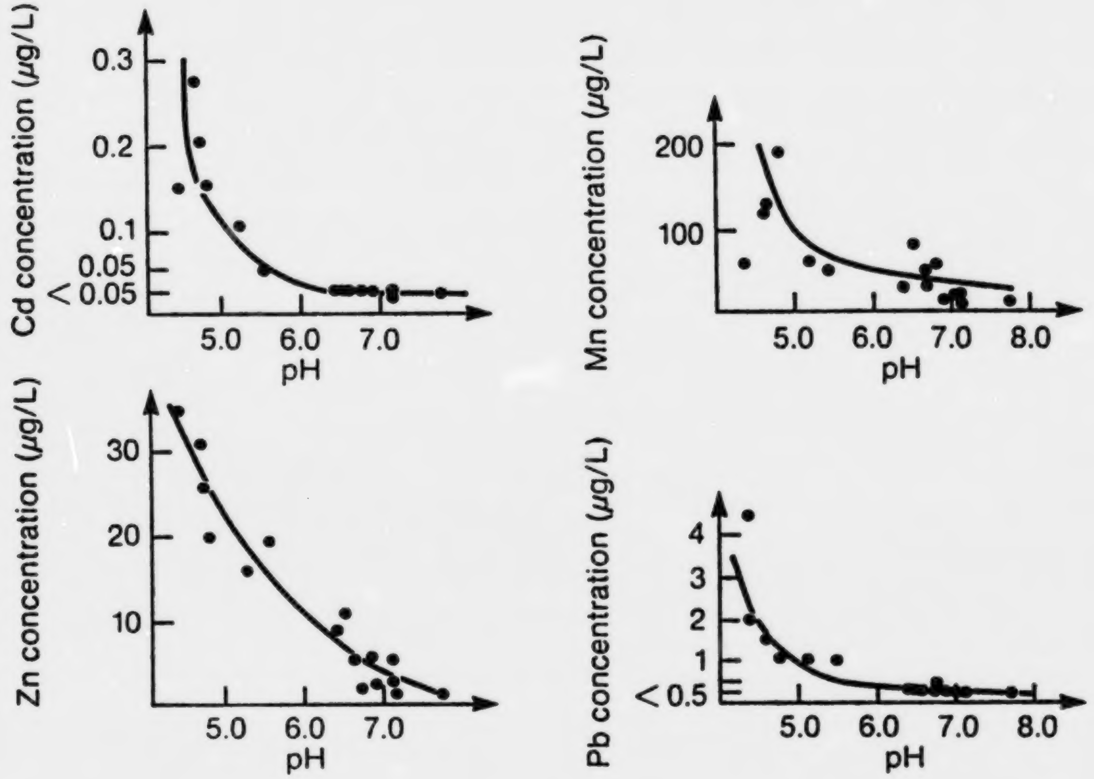

Figure V.3. Metals in 16 lakes on the Swedish west coast with similar metal deposition but with different pH, December, 1978.

Source: Dixon, 1980.

has occasionally been reported (e.g. Heusgem and DeGraeve, 1973); the probable source was lead stearate, which is used in the manufacture of polyvinyl plastics. For the most part, free-running tap water contains much less lead than the first flush, which results from water being held in pipes for some time, e.g. overnight. Isolated cases of human exposure to lead-contaminated drinking water in the US Adirondacks have been found, which would result in a lead concentration in the blood as high as 59 µg/dL (Moskowitz et al., 1986).

Flushing the system is appropriate to get rid of lead mobilized by acidic water, but it does not work for another source of lead in drinking water. Roof catchment cistern systems are still in use in certain areas, e.g. western Pennsylvania, Ohio and parts of the Canadian Maritimes and Prairies. In such systems, the lead comes mainly from atmospheric deposition, which washes from the roofs and accumulates in the cistern, largely as sediment. In rural areas with systems having no safeguards to prevent the suspension of lead in cistern water, drinking water can exceed the standard of 50 µg/L for lead. In a study by Young and Sharpe (1984) for Pennsylvannia, 9 out of 40 roof catchment cistern systems exceeded this standard. In Canada, there are roof catchment cistern systems in the Maritimes and in parts of western Canada. The Commission is unaware of any recently reported problems from these areas, or of systematic studies that have been done in Canada for roof catchment cistern systems.

Rural dwellers and cottagers using lake water as a source of drinking water may be comforted by bacteriological tests showing that their water is safe. However, lead is not usually measured in drinking water; if acidified surface waters are used for drinking, the potential exists for concentrations of lead and other metals to be quite high. Again, running the tap will not solve this problem.

Unfortunately, quantitative data relating lead in drinking water to acidic deposition are lacking, though there are extensive data on lead in drinking water. However, the general impression from unpublished data is that the distribution system represents the major source of risk for lead in drinking water (Norton, 1985).

(b) Lead in Fish in Acidic Waters

It has been recognized for decades that the concentration of mercury in fish muscle shows an inverse relationship with the pH of the surface water, even in the absence of demonstrated mercury pollution (Lindqvist et al., 1984). Certain other metals, which may increase in concentration in acidic water, have recently been considered. Body burdens of lead in fish may show, as does mercury, an inverse relationship with water pH (Wiener, 1983; Suns, 1985). Furthermore, laboratory studies have shown increasing accumulation of inorganic lead by fish with decreasing pH (Merlini and Pozzi, 1977; Hodson et al., 1978a). However, unlike mercury, lead does not concentrate in the edible fish flesh (axial muscle tissue), but rather in tissues and organs not generally consumed by man, such as kidney, liver and bone (Hodson et al. 1978b; Kumada et al., 1980; Fjerdingstad and Nilssen, 1983). Therefore, lead levels in fish, though an indication of some alteration of the biogeochemical cycling of lead as the result of acidification, are not perceived as a health risk to man. Acidification of surface waters has also not been demonstrated as causing any lead-related effects on fish or other aquatic biota at the present time.

5. Impact of Acidic Deposition on Exterior Surfaces

All painted surfaces weather, and the chemical and physical changes involved are well-known (Ter Haar and Aronow, 1974). The corrosive effect of acidic deposition on outside surfaces containing leaded paint may accelerate the process (Malone, 1984). This may transfer lead from painted surfaces to soils and dusts, where children may come into contact with it. No attempt appears to have been made to quantify this process.

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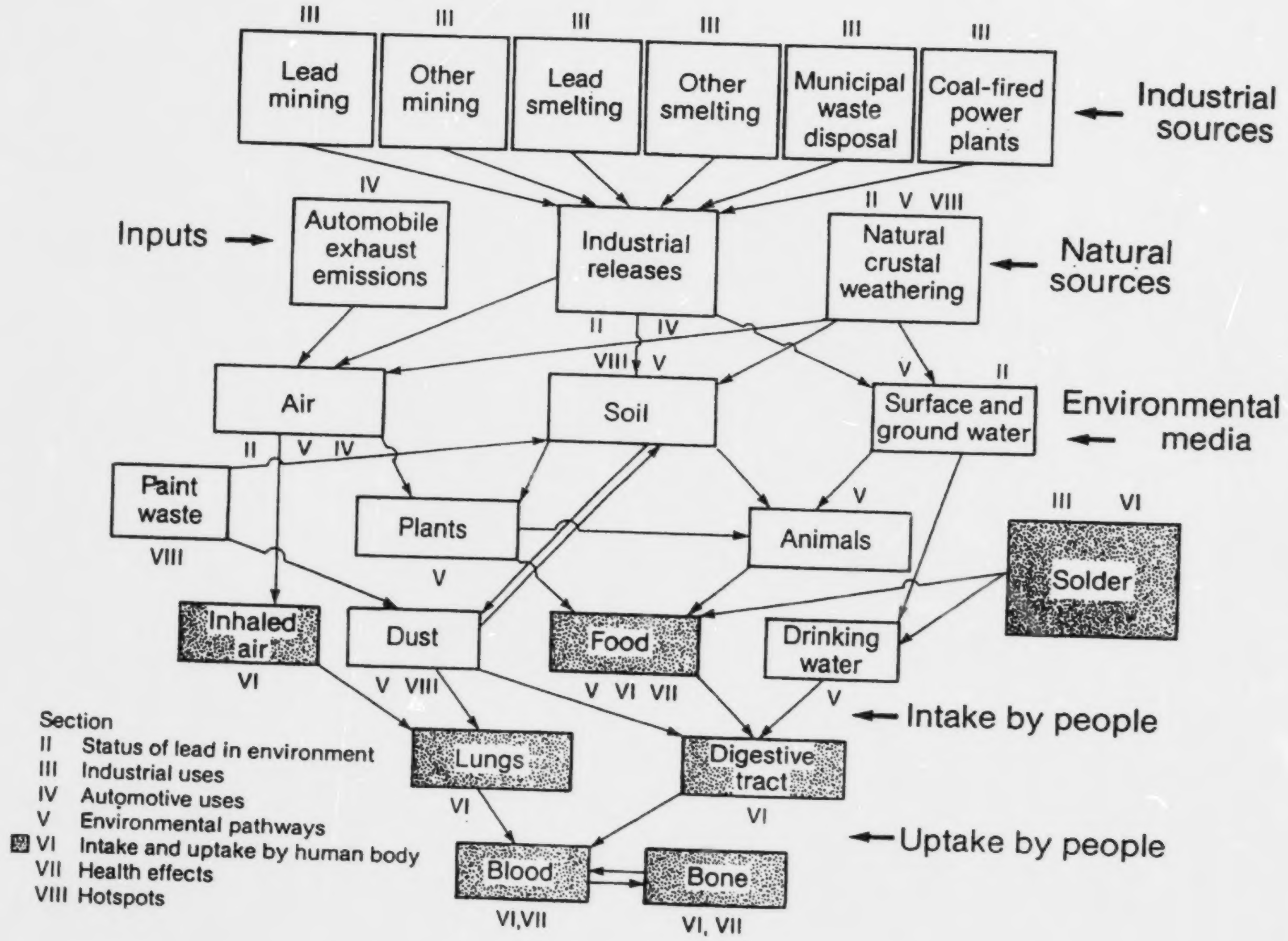
Shading indicates specific
sections (numbered) of text



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Lead mining
III
Other mining
III
Lead smelting
III
Other smelting
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Industrial sources
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Inputs
Automobile exhaust emissions
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V Environmental pathways
▨ VI Intake and uptake by human body
VII Health effects
VIII Hotspots

Area covered in Section VI

VI Intake and Uptake of Lead by the Human Body

This section deals with the last two rows of 'boxes' in Figure I.1 (see Section I), i.e. with the intake of lead into the body, and its uptake by cells and tissues. The main routes of entry into the body (intake) are inhalation and ingestion. Organic compounds of lead may be absorbed through the skin, but this is not an environmental hazard to the public because the concentrations of organic lead in ambient air are very low. This latter route will not be considered further. The effects of lead after uptake, and the bodily processes involved, are considered in Section VII.

The most readily available index of human exposure to lead is the concentration of blood lead (PbB, µg/dL), though this may not always reflect potential toxicity, especially in young children (see Section VII,. Uptake/blood lead relationships are complicated processes, but for convenience can be considered in two stages:

- the relationship between PbB and rate of uptake of lead (T, µg/d) into the bloodstream. This depends on age and sex, and on the period over which uptake has continued at the stated rate. There is much debate on whether PbB at equilibrium is linearly related to T, i.e. whether an increase or decrease in T induces a proportional change in PbB. It is important to determine this relationship over the range of T and PbB encountered in real-life situations.

- the relationship between T and the amounts of lead inhaled or ingested. For inhalation, the main factors are the ventilation rate of the individual (m^3/d), which depends on age and physical activity, and the fractional deposition of the inhaled aerosol in the lung. For small particles (less than about 1-μm diameter), which comprise most of the urban aerosol, experiments have shown virtually complete absorption of the lead deposited in the lung. Larger particles, e.g. some smelter emissions and resuspended dusts, tend to be deposited in the upper respiratory tract, from which they are removed by mucociliary action and swallowed. Absorption is then usually less than 50%. For ingestion, the relationship between intake as food or drink and uptake from the gut is much more variable, depending on a number of dietary factors. When lead is taken in aqueous solution without food, uptake from the gut may be as high as 80%, even in persons of good dietary status; however, when lead is taken with a full meal, uptake by adults is usually less than 10% (Rabinowitz et al., 1980; Flanagan et al., 1982; Heard and Chamberlain, 1983; James et al., 1985). This difference in fractional uptake is important when comparison is made of the contributions of lead in food and in water, other beverages or dust that may be ingested without food. Balance studies (Ziegler et al., 1978) have indicated that infants absorb proportionately more lead than do adults following oral intake, and this also seems to be the case for animal experiments (Kostial et al., 1971; Forbes and Reina, 1972).

1. Sources of Information

In the last 20 or 30 years, great efforts have been made in several countries to quantify the relationship between blood lead and dietary or airborne lead. The studies have been of three main types: epidemiological studies, human experiments and animal experiments.

(a) Epidemiological Studies

Surveys have been made of PbB in populations exposed to different levels of environmental lead, e.g. urban versus rural residents, children living near smelters versus those more distant, and communities with water supplies high in lead versus those with low lead.

These studies have the advantage that they deal with people of both sexes and all ages in real situations. But there are also difficulties in interpretation, especially with respect to the isolation of the effects of the various environmental variables. Three of the most important variables, i.e. lead in air (PbA, $\mu g/m^3$), lead in house dust (PbD, µg/g) and lead in soil (PbS, µg/g dry weight), tend to be high in urban areas and low in rural areas. There are statistical techniques for isolating the effects of specific variables, but the outcome is often that only a small part of the variance in PbB can be attributed to environmental factors. Furthermore, the maintenance of strict control on sampling and analytical quality is more difficult in field than in laboratory work.

(b) Human Experiments

Volunteer subjects have been exposed to elevated levels of lead in air or diet, in some cases over periods of months or years (Kehoe, 1961; Griffin et al., 1975). In such studies, sufficient lead has to be given to elevate the blood lead of the subject to a certain degree, in order to obtain a measurable result. However, if stable or radioactive isotopes of lead are used (Rabinowitz et al., 1976; Chamberlain et al., 1978), the uptake into the body of a small dose, its persistence in blood and its excretion can be followed irrespective of the previous lead burden of the individual. The disadvantages of the experimental approach are that volunteers are usually male, adult and healthy, which of course is not a typical sample of the general population. Also, the period over which the experiments are done is necessarily short compared with the time (years) needed to attain equilibrium of lead concentration in body tissues.

(c) Animal Experiments

The uptake and metabolism of lead by animals, including primates, differ from those of man, so that only limited use can be made of animal experiments. However, it is sometimes useful to conduct animal experiments, e.g. as elaborate mechanisms to be used to extrapolate from the adult to the child or infant.

2. Synthesis of Experimental and Epidemiological Data

Goldsmith and Henter (1967) were the first to attempt to draw together experimental and epidemiological data for lead. They obtained a regression of log PbA against log PbB, over the range of PbA from 1 to 3 $\mu g/m^3$:

$$\text{PbB } (\mu g/dL) = \text{constant} + 1.7 \text{ PbA } (\mu g/m^3)$$

In other words, blood lead increased by 1.7 $\mu g/dL$ for each increase in air concentration of 1 $\mu g/m^3$. Many other such regressions have been derived, mostly giving slopes between 1 and 3 $\mu g/dL$ per microgram per cubic metre.

A more fundamental approach is to relate PbB to the amount of lead absorbed into the bloodstream, uptake, T ($\mu g/d$). Studies with radioactive tracers have shown similar metabolism of inorganic lead regardless of the route of entry (lung or gut). Figure VI.1 shows a summary derived by EPA of the relationship between PbB and T. Clearly, there appears to be good agreement among different authors. From the figure, it is also apparent that the same relation holds for adults and children:

$$\text{PbB } (\mu g/dL) = 0.4 \text{ T } (\mu g/d)$$

This relationship applies at equilibrium, which may take several years to attain, because PbB depends on resorption of stored lead from bone as well as on current uptake (Chamberlain, 1985). Several surveys have indicated, moreover, that the relationship between PbB and T is not straight, but curvilinear, with decreasing slope as PbB increases (NAS, 1972; Chamberlain, 1985).

(a) The Inhalation Route: Estimation of Lead Uptake

A person doing light manual work inhales about 20 m^3 of air per day, but for the average city dweller 15 m^3/d is more usual. Experiments have shown (Kehoe, 1961; Chamberlain et al., 1978) that about 50% of submicrometre-sized particles are deposited in the lung and absorbed into the bloodstream. Assuming 50% retention and 15 m^3/d ventilation, T = 7.5 PbA. Using Figure VI.1, this gives a slope of 0.4 x 7.5 = 3 μg/dL per microgram per cubic metre for the relationship between PbB and PbA. This tends towards the higher end of the rates found in epidemiological and experimental studies. However, most of the latter were not continued sufficiently long to approach equilibrium between PbB and PbA.

The Isotopic Lead Experiment (Facchetti and Geiss, 1982; see Appendix V) showed that 25% of the lead in blood of Turin adult residents derived from

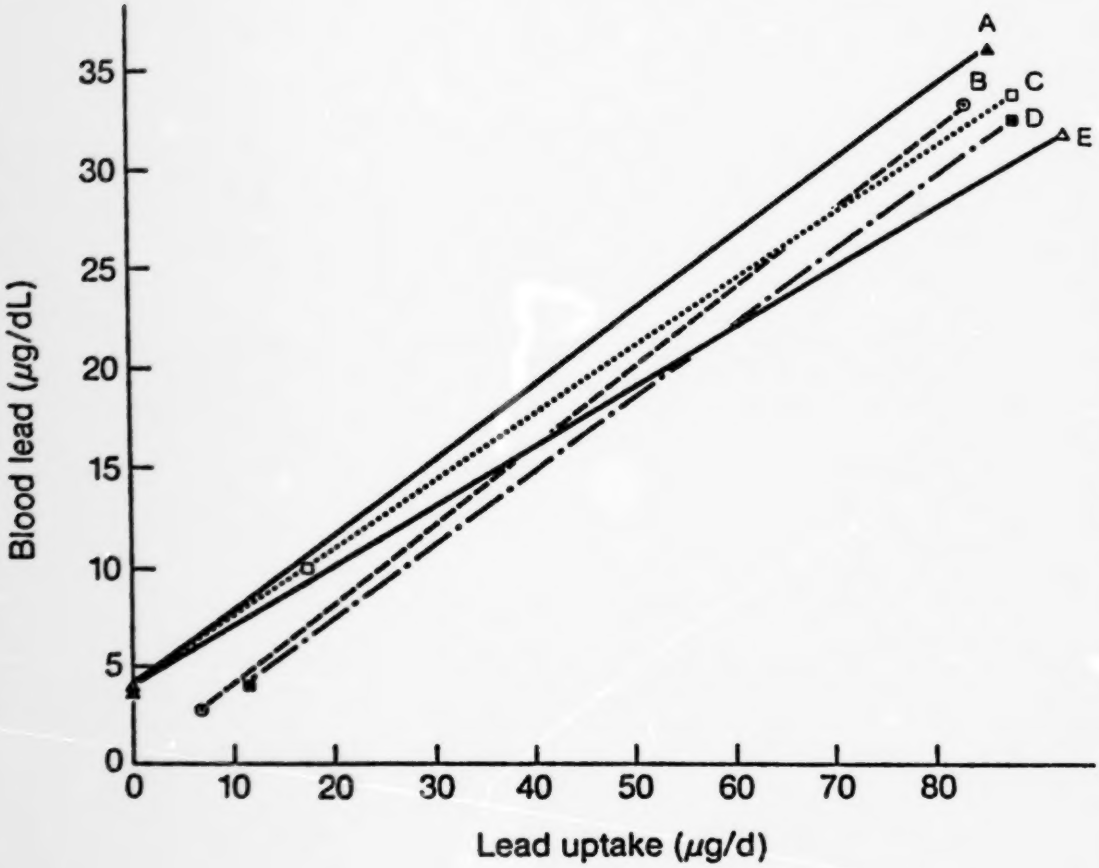

Figure VI.1. Summary of relationships between daily lead uptake and blood lead, derived from: (a) Ryu et al. (1983) — curves A (▲) and E (△) represent estimates using 0.42 and 0.53 for lead absorption rates through the gut; (b) Harley and Kneip (1985) — curve B (⊙) represents estimates for 2-year-old children; (c) Chamberlain and Heard's (1981) analysis of epidemiological data on adult men (curve C); and (d) Rabinowitz et al.'s (1976) metabolic study on adult men (curve D; ■).

Source: EPA (1986)

gasoline lead. The mean adult PbB in Turin was 22.5 µg/dL and mean PbA about 1.7 µg/m^3. These results are consistent with a PbB/PbA slope of 0.25 x 22.5/1.7 = 3.3 µg/dL per microgram per cubic metre. Because the dust in Turin also showed an isotopic shift, the pathway via street dust would be included in this result. The isotopic ratio in blood of Turin schoolchildren showed a somewhat greater shift than did the ratio in adults, but PbB was lower in children, and the derived PbB/PbA slope the same as for adults.

Some comments on the Turin experiment are appropriate at this point. For meteorological reasons, and because the traffic was very heavy and the lead concentration in gasoline relatively high (0.6 g/L), PbA in Turin was higher than in most cities. Typical PbA levels (circa 1980-1985) in North American and European cities are 0.5-1 µg/m^3 in inner cities and 0.1-0.3 µg/m^3 in outer cities and rural areas. With a PbB/PbA slope of 3 µg/dL per microgram per cubic metre, comparisons of inner-city and rural populations would give a difference of 1 or 2 µg/dL between the two populations, taking account only of the inhalation route. In Britain in 1979, the difference in adult PbB between inner- and outer-city populations averaged 1.8 µg/dL (Southwood, 1983). A recent survey of PbB in 5-year-old children and their mothers from central London and rural Suffolk, UK, gave the results shown in Table VI.I.

The difference in PbB between urban and rural residents was not significantly different for mothers or for children. The ratio of 1.2 or 1.3 for children to mothers was similar to that found in several other recent surveys (Chamberlain, 1983). The percentage distributions of blood lead for children in urban versus suburban Ontario (Ontario Ministry of Health, 1985) are quite similar, with a difference of 2.1 µg/dL between the means for the two groups. This compares very closely with the data for UK adults (Southwood, 1983).

Table VI.I

Comparison of Blood Lead Between London and Suffolk Mothers and Children, 1983-1985

	PbB (µg/dL)			Lead in dusts (µg/g), means	
City	Mothers	Children (5 years)	(Ratio, children/mothers)	Road dust	Home dust
London	6.8	7.9	(1.2)	1880	927
Suffolk	5.1	6.5	(1.3)	144	333

Source: Strehlow and Barltrop, 1986

(b) The Ingestion Route: Estimation of Lead Uptake

As previously explained, uptake of lead from the gut is variable. From Kehoe's (1961) work, a figure of 10% uptake has been derived, but this does not take account of the fact that some faecal lead is endogenous in origin (Chamberlain et al., 1978). Also, Kehoe's subjects took their lead doses with meals, which would minimize the uptake. To make a conservative estimate, a figure of 15% uptake will be assumed. This gives T = 15 µg/d for an intake of 100 µg/d. From Figure VI.1, PbB should increase by 0.4 x 15 = 6 µg/dL for each 100 µg/d of dietary intake in adults.

In several European countries, lead in drinking water is still an important source for humans, though strenuous efforts are being made to decrease lead from this source. Figure VI.2 shows correlations of blood lead with water lead from several surveys. In the UK, the average adult consumption of drinking water is estimated to be 1.2 L/d. Hence, it would be expected that PbB would show an increase of 1.2 x 6 = 7.2 µg/dL for each 100 µg/L in drinking water. This is not inconsistent with the epidemiological surveys.

The amount of lead in human diet has decreased in recent years, largely by elimination of lead-soldered cans (Dabeka, HWR). Dabeka's most recent estimate of adult dietary lead intake in Canada is 54 µg/d; assuming 15% uptake and the relationship from Figure VI.1, food should contribute only about 3 µg/dL to PbB. Such low levels have not yet been recorded, but it may be a decade or more before reductions in intake of lead are fully reflected in blood lead. Manton (1985), by analysis of stable lead isotopic ratios in blood, diet and bone, estimated that more than half of adult blood lead may be derived by resorption from bone. If the fractional absorption of lead from

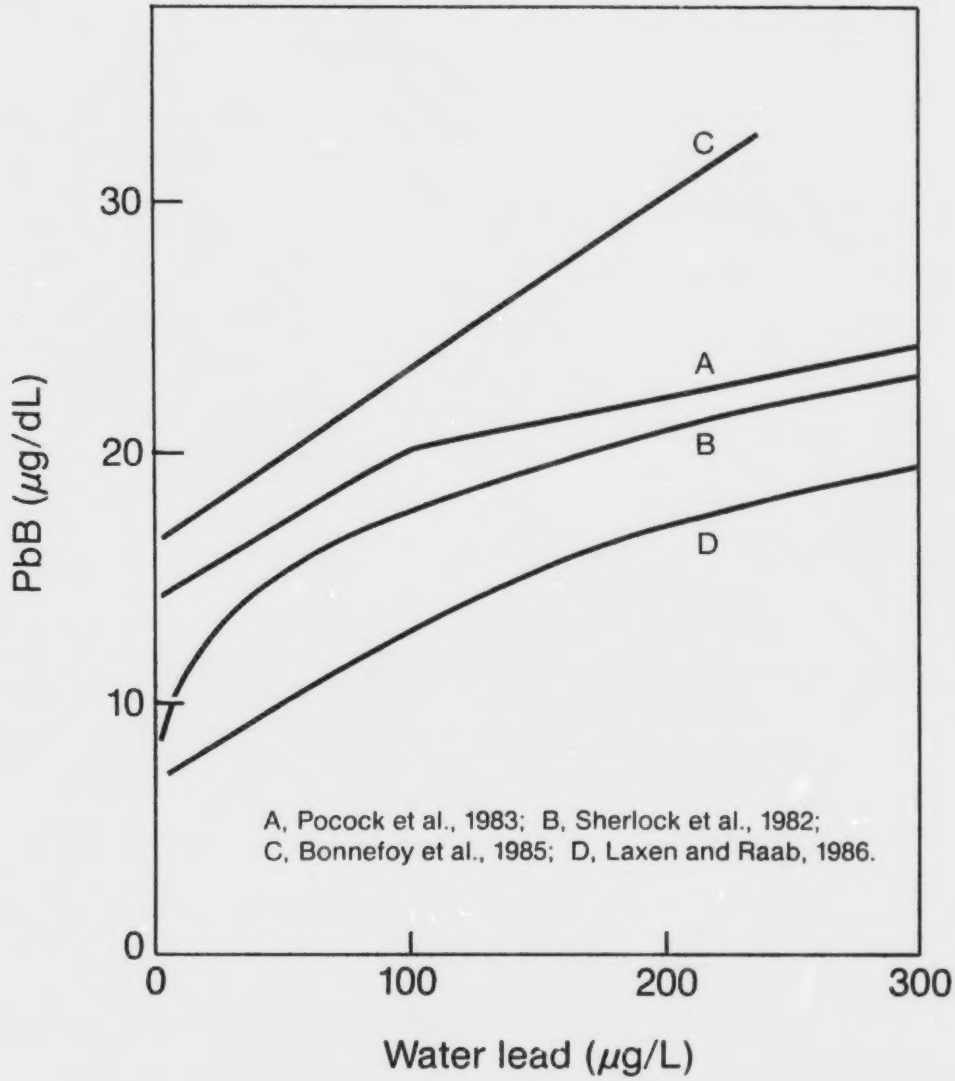

Figure VI.2. Estimated effect of water lead on blood lead.

the gut of young children is much higher than that of adults, this should be reflected in the PbB levels (though a large fraction is deposited in their bones). Sherlock et al. (1982) obtained the following regressions for the relation between PbB and intake of lead:

$$\mathrm{PbB} = -1.4 + 18.9\ I^{1/3}\ \text{(adults)}$$

$$\mathrm{PbB} = 2.5 + 26\ I^{1/3}\ \text{(infants, bottle-fed)}$$

where I is the weekly intake in milligrams. At the 2 mg per week level, these regressions indicate that PbB of infants is about 50% higher than that of adults for equal intake (not normalized for body weight), which is considerably less than would be expected from the balance study of Ziegler et al. (1978).

(c) Direct Ingestion of Dust or Soil

This is the most difficult route to evaluate, and one of great potential importance, especially for children. Several studies have shown correlations between PbB in children and the amounts of lead on their hands (Sayre et al., 1974; Roels et al., 1980), and higher PbB in children who do not wash their hands before snacks or meals compared with those who do (Sherlock et al., 1985).

In EPA models, it is assumed that a 2-year-old child ingests 100 mg of soil per day. If the lead content of soil (PbS) exceeds 500 µg/g dry weight--as it does in most of central London, UK (Thornton, PWR)--then an intake of 100 mg/d of soil would include 50 µg/d or more of lead. This would be the major source of lead intake for the young child. However, Table VI.I indicates that this does not show up statistically for 5-year-old children, because the excess PbB in the urban population with very high lead in dust is the same for children and their mothers. In earlier work in Derbyshire, England, where soils contain up to 30 000 µg/g of lead as a result of old mining activities, Barltrop et al. (1975) found a regression of PbB (2- to 5-year-old children) on PbS equivalent to only 0.6 µg/dL per 1000 µg/g in soil.

By comparison, if the child ingests 100 mg/d, if gut uptake is 45% (as suggested for infants) and if the linear relation of Figure V.1 applies, then the PbB/PbS slope should be 0.1 x 0.45 x 0.4 x 1000 = 18 µg/dL per 1000 µg/g in soil. If that were applied to London children, then their PbB levels should be three times higher than those of their country cousins, which is clearly not shown in the data. In another British study, PbB was compared in people living in Shipham, Somerset and a control village, North Petherton. Shipham was investigated because soil cadmium levels were very high, but PbS was high also, averaging 3800 µg/g in Shipham compared with 300 µg/g in North Petherton (Southwood, 1983). No significant difference in PbB of adults or children was found in Shipham compared with North Petherton.

The problem can be further illustrated by comparison of data for central London, UK, and South Riverdale in Toronto. In London, industrial emissions of lead are slight (except in one or two localities), but there is a legacy of lead in soil from industrial and domestic use over the centuries. In South Riverdale, industrial emissions to the atmosphere are comparatively recent, and still continue, though they have been reduced over the past 7 years (see Section VIII). Table VI.II illustrates the results.

Table VI.II

Lead in Air, Fallout, Soil and Blood

	South Riverdale	Reference	Central London, UK	Reference
PbA (in 1985) ($\mu g/m^3$)	0.43	(a)	0.5-1	(b)
Lead fallout (mg/m^2 per month)	40	(a)	5-10	(b,c)
PbS ($\mu g/g$)	1000	(a)	670	(d)
PbB (4- to 6-year-old)	15.5	(a)	8-10	(e)

Sources:
(a) South Riverdale Community Health Centre, 1985; 1986
(b) Duggan and Barton, 1983
(c) Harrison et al., 1975
(d) Thornton, PWR
(e) Table VI.I and Sherlock et al., 1985

It appears that environmental conditions in South Riverdale and in central London are fairly similar, except for the rate of lead dust-fall, which is much higher in South Riverdale. Arguing from the results of Brunekreef et al. (1983), Chamberlain (PWR) suggested that there is a regression of PbB on the rate of lead fallout (PbF, $mg/m^2/d$) with a slope of 5, i.e. PbB increases by 5 µg/dL for each milligram per square metre per day of dust-fall. When this regression is applied to the South Riverdale situation, the results are as shown in Table VI.III.

Thus, the effect of PbF on PbB, derived from the work of Brunekreef et al. (1983), would more than account for the elevation of PbB in South Riverdale, both past and present, compared with other urban areas. A regression of PbB on PbS (i.e. on the cumulative lead in the soil instead of the rate of fallout) would be less successful, because PbS has presumably increased since 1972 (except where soil has been removed; see Section VIII), whereas PbF has decreased since 1972, though it is still high compared with normal urban areas. It is the current rate of fallout, not the cumulative fallout of lead, that is most important. The current fallout is mobile, enters houses and motor vehicles and is easily resuspended.

3. Estimated Daily Exposure to Lead

So far, we have avoided giving absolute values for the intake or uptake of lead in man. Indeed, it is clear from the various uncertainties expressed in the foregoing exercise that such values may be misleading. Nevertheless, it is desirable to have a picture of the relative contribution of each source to the total daily uptake.

Table VI.III

Fallout and Children's Blood Lead in South Riverdale

Year	Rate of fallout PbF (mg/m^2/d)	Contribution to PbB (µg/dL) assuming regression	Recorded PbB
1972	6	30	24
1985	1.2	6	15.5

Source: Derived from Rachlis
and Hertzman, 1986

Reasons for attempting to do this include the need to use resources most effectively in controlling the various sources of lead, and the need to regulate lead exposure as closely as possible. A number of models have been produced to address these questions. EPA (Elias, PWR) provides a fairly detailed account of intake and uptake of lead, and a more site-specific model is provided in Report VI (by ESSA Consultants) for various regions in Toronto. It should be recognized that such models give at best only rough estimates of exposure. If one adds the uncertainties of the uptake (T) and PbB relationships to the uncertainties of uptake and intake, the task appears overwhelming. Nevertheless, we consider that these estimates can be useful, and provide, in Table VI.IV, HWC's most recent estimates for adults and children. Though food provides a major proportion of uptake for both adults and children, there are clear differences with respect to the relative contribution from air and soil for adults and children, respectively.

These calculations predict blood lead values that compare quite well with those observed during the Ontario blood survey among children in 1984 (Ontario Ministry of Health, 1986).

Table VI.IV

Balance Scheme for Intake and Uptake of Lead

Parameter	Urban[f] Adults	Urban[f] Children (1-3 years)	Rural Adults	Rural Children (1-3 years)
1. Average air lead concentration ($\mu g/m^3$)[a]	0.46	0.46	0.10	0.10
2. Intake of lead from air[b,*]	6.8	2.1	2	0.6
3. Total lead uptake from lungs (µg/d)(40% of line 2)	2.7	0.8	0.80	0.24
4. Median tap water lead concentration (µg/L)[c]	2	2	2	2
5. Intake of lead from tap water (µg/d)(line 4x1.5 or 0.6)[d]	3	1.2	3	1.2
6. Intake of lead from food (µg/d)[e]	71.5	37.4	71.5	37.4
7. Total intake of lead from water and food (µg/d)	74.5	38.6	74.5	38.6
8. Lead uptake from water and food in gut (µg/d)[d]	7.5	19.3	7.5	19.3
9. Average lead concentration in dust and dirt (µg/g)[g]	850	850	200	200
10. Amount of dust digested (g/d)[h]	0.02	0.1	0.02	0.1
11. Intake of lead from dust and dirt (µg/d)	17	85	4	20
12. Lead uptake from dust and dirt in gut (µg/d)[i]	1.7	25.5	0.4	6
13. Total lead uptake from lungs and gut (µg/d)	11.9	45.6	8.7	25.5
14. Percentage daily lead uptake from air	23	2	9	1
15. Percentage daily lead uptake from water	3	1	3	2
16. Percentage daily lead uptake from dust/dirt	14	56	5	24
17. Percentage daily lead uptake from food	60	41	83	73
18. Predicted blood lead conc. (µg/dL)**	6.4	10.3	4.5	7.6

[a] For urban residents, twice the 1982 annual geometric mean concentration from NAPS samples was used, because it is thought that the NAPS data underestimate human exposure (see text).

[b] Standard volume of air inhaled per day: adults - 20 m^3; children - 6 m^3.

[c] Meranger, 1979, 1981; Canadian Duplicate Diet Study (Food Directorate, HWC, 1986).

[d] Volume of tap water consumed: adults - 1.5 L/d; children - 0.6 L/d.

[e] Value based on the average total diet for males and females and for 1-4-year-olds (Food Directorate, HWC, 1986).

[f] In 1981, approximately 75% of Canada's population lived in urban communities.

[g] Arithmetic mean of house dusts, Toronto (Roberts et al., 1974). Rural concentrations found to be one quarter of urban levels (Ontario Ministry of Health, 1985).

[h] NAS, 1980.

[i] Percentage of lead in dust and dirt absorbed by children estimated to be 30%.

* Urban indoor lead concentrations approximately 70% of outdoor levels. Time spent outdoors (15%) 3.5 hours; indoors (85%) 20.5 hours.

**Using relationship between uptake of lead and BPb given by Southwood (1983) for adults; and by Drill et al., 1979, for children.

Source: Health and Welfare Canada. 1985;1986

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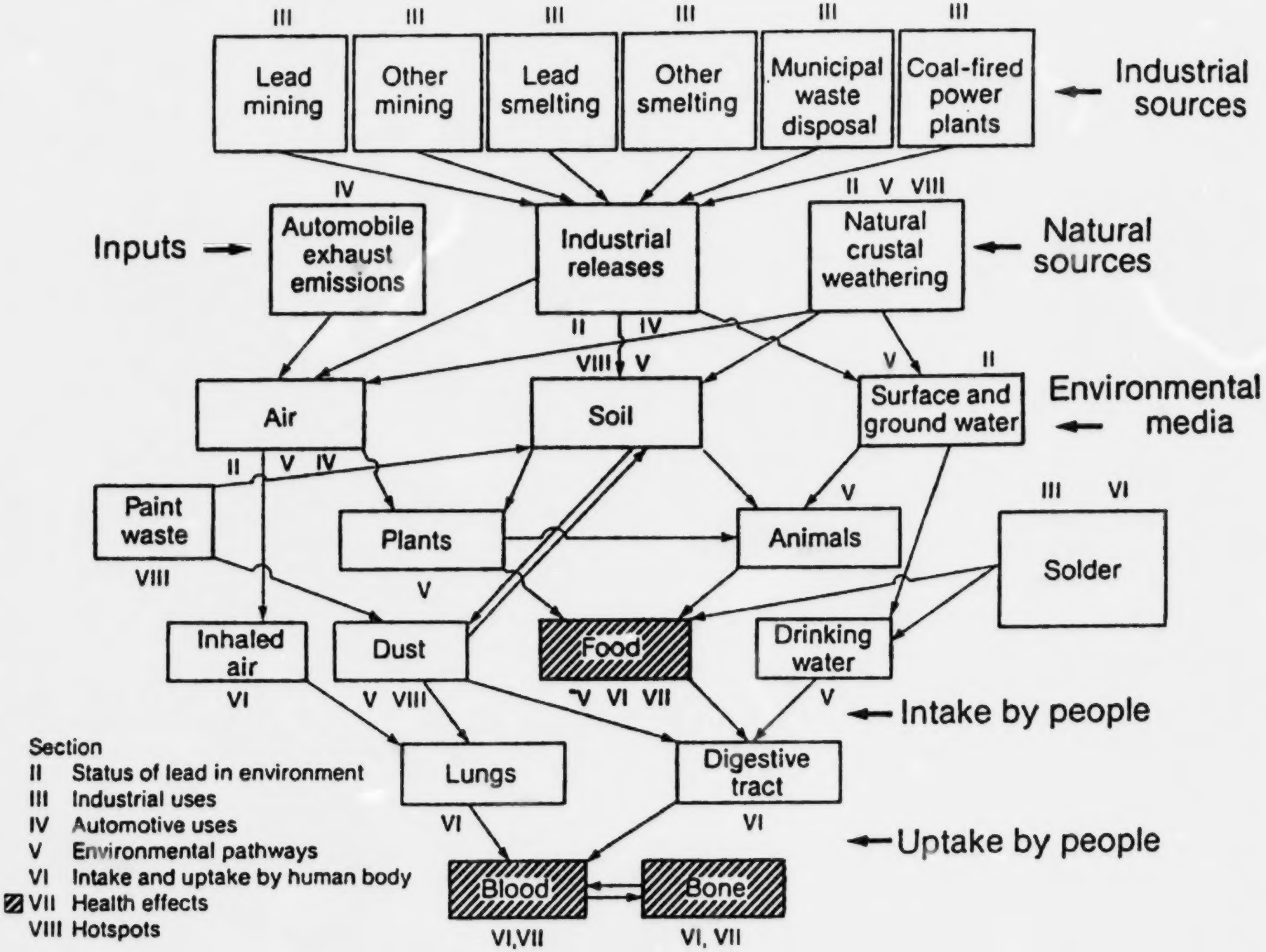
Shading indicates specific
sections (numbered) of text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Other mining
Lead smelting
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Municipal waste disposal
Coal-fired power plants
Industrial sources
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Inputs
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Dust
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Food
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Drinking water
V
Intake by people
Lungs
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Digestive tract
VI
Uptake by people
Blood
VI,VII
Bone
VI, VII
Section
II Status of lead in environment
III Industrial uses
IV Automotive uses
V Environmental pathways
VI Intake and uptake by human body
▨ VII Health effects
VIII Hotspots

Area covered in Section VII

VII Health Effects of Lead Exposure

Having reviewed the intake and uptake of lead by the human body, we now turn to the effects of this lead on health, in the broad sense of that term.

As mentioned earlier in the report, there is no doubt that lead is a dangerous toxin at high levels of uptake and body burden. Such high levels are rare in the Canadian population. Our discussion will hence focus on the problem: what are the effects, if any, of uptakes typical of this country?

The Commission has examined this problem by trying to answer a series of intertwined questions:

- What are the levels of lead in the Canadian population and how do they compare with those in the rest of the world?
- What are the known or suspected effects on health that one can expect at such levels?
- Which groups of the Canadian population are most at risk?
- Which are the target organs and systems most likely to be affected?
- What are the consequences of the present situation for Canadians and what can be done to improve them?

Answers to these questions have been sought by the Commission, not only through a comprehensive review of the investigations reported in the literature (and at the several workshops held by the Commission), but also by a critical assessment of the designs and methodologies used in published studies.*

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* Four US researchers whom we have cited differ from us in their conclusions. Their views are presented in Appendix XI.

1. Effects of Low Lead Exposure

Lead is a toxin affecting several functions in the human organism. Its effects depend on many factors, some genetic (such as individual or racial susceptibility), some acquired (nutritional status, socio-economic situation) and some depending on the chemical form of the lead (organic or inorganic) and the quantity and duration of exposure. Some of these factors are well-known, but the role of others remains unclear. Most effort to clarify their role has, of necessity, been directed to animal experimentation. The Commission's review of the relationship between lead and health has placed emphasis on human studies, referring to animal experiments whenever they help our understanding of the mechanisms involved in the toxicity process.

2. Factors Determining Absorption and Retention of Lead*

The absorption of lead by the human body depends on many factors. Children tend to absorb more than adults, particularly through the intestinal tract. Because of children's rapid growth, nutrients are transformed into living matter and energy much more quickly than in adults (i.e. children have a higher metabolic rate), and children absorb and retain more lead than do adults. A representative study in infants estimates that 41.5% of lead ingested is absorbed and 32.6% is retained (Ziegler et al., 1978). This compares with an estimated 5-15% in adults (Kehoe, 1961; Rabinowitz et al., 1975, 1980; Chamberlain et al., 1978). Moreover, children's lead burden (i.e. the total amount of lead in the body) appears to be more active biologically; the proportion of lead deposited in bone is lower than in adults (65% compared with 90%; Barry and Mossman, 1970) and is more easily mobilized (i.e. put back into circulation in the blood). The result is that there is proportionally more lead in the soft tissue in children than in adults. This is in part due to the growth process, through which children constantly mobilize bone mineral during the physiological remodelling of bone (Rabinowitz et al., 1976, 1977; Chamberlain et al., 1978; Rosen and Markowitz, 1980).

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* This discussion continues the treatment of uptake in Section VI, and considers the physiological and biochemical processes involved.

Rosen (HWR) summarized the greater metabolic liability of children compared with adults. Children absorb considerably more dietary lead than do adults. This increased overall absorption is accompanied in children by a decreased ratio of urinary excretion to absorbed lead, with a consequent increased retention of lead. Because the concentration of lead in soft tissues in children (though proportionately greater than in adults) is not particularly high, there results a large pool of mobilizable bone lead. This makes blood lead level a poor index of body burden in children though it is the best available.

There is also evidence that the sexes may differ as regards absorption of lead, though differences of intake and uptake exist because of variations in cigarette smoking and alcohol consumption. Most studies have reported little or no difference in blood lead levels between sexes. Where these have been found, they have usually been attributed to greater exposure to lead on the part of the male. Stuik (1974), however, in a controlled study on volunteers aged 18-26 years, demonstrated that females respond to lead with a greater increase in erythroporphyrins; a similar response was found for non-pregnant female workers (Roels et al., 1979). Among the hypotheses proposed to explain these findings are iron deficiency (see below) and the role of testosterone.

Dietary components are another major factor in determining lead absorption (and possibly toxicity). Interaction between lead and nutrients modifies the proportion of lead absorbed from the gastrointestinal tract, its distribution in the body and its toxicity at the cellular level. For example, in adults a greater amount of lead is absorbed by an empty stomach than if the lead is mixed with food (Rabinowitz et al., 1980; Heard and Chamberlain, 1982; Blake and Mann, 1983). We have not seen comparable studies with children.

Calcium and phosphorus appear to play important roles as dietary components. Low calcium levels have been found associated with a greater prevalence of elevated lead levels in both children and adults. Calcium and phosphorus were found to be inversely related to lead absorption and retention in children (Ziegler et al., 1978). Barton et al. (1978) found that calcium in the intestinal tract reduced absorption of lead. The exact mechanism by which the presence of calcium decreases the absorption and toxicity of lead remains to be elucidated (Goyer, 1978), but there is little doubt that low dietary calcium can increase the mobile fraction (i.e. the fraction that can easily be released into the bloodstream) of lead in the body and its potential toxicity.

The exact role of this action is, however, difficult to interpret because of the complexity of the human diet (Johnson and Tenuta, 1979) and because socio-economic and racial groups that consume diets lower in calcium also have potentially higher exposures to environmental lead (Mahaffey et al., 1982).

The role of iron in lead absorption under typical dietary conditions is not clear (Watson and Hume, 1983). Most of the evidence indicating an increase in tissue lead levels when body iron stores are reduced comes from animal experimentation (Mahaffey-Six and Goyer, 1972; Ragan, 1977; Barton et al., 1978; Hamilton, 1978; Flanagan et al., 1979). Inconsistent results were obtained by Watson et al. (1980) and Flanagan et al. (1983) on iron-deficient human adults. It is not at present clear whether these different results reflect methodological differences, the degree of iron deficiency or a more basic species-related difference (Mahaffey, 1985). The same author also pointed to the public health importance of increased lead toxicity to iron-deficient persons, because iron deficiency is one of the most prevalent nutritional deficiencies in all parts of the world. The interaction of lead with copper and iron must also be considered (Klander and Petering, 1977), because both copper deficiency and excess lead can independently interfere with iron metabolism, and lead may in turn interfere with the metabolism of copper, leading to dysfunction in iron metabolism.

A further interaction at the heme synthesis level has been proposed by Finelli et al. (1975), because zinc is required for the synthesis of ALA-D (see below, p. 192 et seq.). Experiments in rats had indicated that administration of excess zinc in high-lead diets was associated with a decrease in the toxic effects of lead in the blood (hematopoietic) system (Thawley et al., 1977). Zinc appears to impair lead absorption from the gut (Cerklewski and Forbes, 1976). The association between low zinc and elevated lead levels in the body has been found by several investigators (e.g. Markowitz and Rosen, 1981), though in a controlled study of oral zinc supplementation in a group of workers (the control group receiving a placebo), Lauwerys et al. (1983) were unable to determine any difference in the metabolism and toxicity of lead.

More uncertain still is the role of high protein and fat in the diet. There is some experimental evidence that low-protein diets enhance absorption of lead (Mahaffey-Six and Goyer, 1972; Barltrop and Meek, 1975). However, high-fat diets (Barltrop and Meek, 1975; Quaterman et al., 1977) have been reported to cause increased absorption of lead. In rats a similar role has been demonstrated for milk (Kello and Kostial, 1973); however, in this study the diet used was also deficient in iron, a fact that may have contributed to the increased lead absorption.

Vitamins have also been found to play a role in the absorption of lead. In particular, vitamin D appears to increase absorption from the gut (Smith et al., 1978). Again, it is difficult to sort out the specific role played by vitamin D, because of the complex interaction with calcium and phosphorus (Sorrell et al., 1977). A protective effect against lead toxicity has been proposed by Papaioannou et al. (1978).

In summary, both nutritional status and diet play a role in the absorption and toxicity of lead. This role is complex, and animal experimentation results, usually obtained within an artificial isolation, cannot readily be applied to humans. Dietary deficiencies (or excesses) in humans are not usually found for a single specific nutrient (iron in women, and calcium during pregnancy, may be exceptions); furthermore, the effect of diet is frequently confounded with socio-economic and racial characteristics, which are usually associated with different exposures. Diets deficient in certain nutrients (particularly protein, calcium, phosphorus, iron and some vitamins) may lead to increased absorption and retention of lead. Certain groups are therefore particularly at risk with respect to lead toxicity: growing children, females (especially when pregnant) and the less affluent sector of society. It is towards these groups that preventive action must be specifically directed.

3. When Does Actual Harm Occur?

At high blood lead levels, such as are usually found only in work-exposed populations, lead has been shown to affect mostly blood; the nervous system, particularly the central nervous system; the kidneys; and the reproductive systems of both males and females. At the levels typical of the Canadian population, the effect of lead is much more subtle, and the early changes that have been found may not be so consistently associated with lead. Among these, a relationship between blood lead and blood pressure has been postulated and challenged (Harlan et al., 1985; Pirkle et al., 1985). Controversy exists as to the level at which effects on heme synthesis may be considered biologically important, as distinct from clinically recognized, as well as the level at which lead may impair the neurobehavioural functions of children. Effects of lead on the peripheral nervous system at levels below 50 µg/dL have also been found to be inconsistent, and the same is true for its effects (at similar levels) on the reproductive systems of both sexes and on mutagenic activity in lymphocytes. These controversies will be examined more fully later in this section.

The Commission has examined the question of when harm can be said to have occurred, from both theoretical and practical points of view. In the health field, the meaning of "normal" has been subject to scrutiny, and attempts have been made to define it from various points of view. It is almost impossible to neatly separate normal from abnormal in the case of the individuals affected. For every organ and tissue, and with respect to both structure and function, there is a range of variability. The term "normal" can be expressed only in terms of this range, rather than as a hypothetical standard (Ryle, 1948).

The human body, like other living organisms, is continuously called upon to react to physical, chemical and biological insults. The changes that occur in the human systems that are the targets of such insults are part of this adaptation process, and cannot by any means all be considered harmful. In most cases they represent a healthy response showing that the organism is dealing with the aggression. This individual tolerance makes health a dynamic rather than a static concept, and the variability within it is considered the normal range.

Disease ensues when these defences are overwhelmed. It can then be classified in terms of its pathological and histological characteristics (WHO's "International Classification of Disease") or, alternatively, in terms of a perception of the functioning of the individual, assessing the consequences of the disease ("International Classification of Impairments, Disabilities and Handicaps") (WHO, 1980).

Another approach by the World Health Organization sets out four categories of "environmental health criteria" that define the levels of environmental exposure to any agent in terms of both time and concentrations below which, according to present knowledge, certain effects do not occur (WHO, 1973):

- Level I the level of detectable effect, below which neither direct nor indirect effects (including alteration of reflexes or of adaptive or protective reactions) have been observed
- Level II the level at and above which there is likely to be irritation of sensory organs, harmful effects on vegetation,

visibility reduction or other adverse effects on the environment

- Level III the level at and above which there is likely to be impairment of vital physiological functions or changes that may lead to chronic disease or shortening of life
- Level IV the level at and above which there is likely to be acute illness or death in susceptible groups of the population

Though this model could be applied using levels of blood lead (or any other measurement of body burden), the same problem would still exist, in that the distinction between the effects at each level continues to be hard to apply in practice, with the possible exception of Level I. The changes between Levels I and II and between Levels II and III, and particularly the definition of the effects at Level III, still evade easy resolution.

In summary, the currently available systems of classification referred to above do not satisfactorily permit clear definition of the point at which harm occurs. Only when the insult affects a statistically significant proportion of the subjects can the probability of harm occurring be epidemiologically established. Whether or not such a probability is acceptable, is, of course, a political judgement.

4. Biochemical Evidence of Lead Impact at Low Blood Lead Levels

The evidence that led to the EPA actions discussed on pp. 7 and 89, and to less vigorous responses in other countries, has come from a variety of studies. Much of this research has been in fields where the existence of lead impacts at blood levels above 30 µg/dL is well established. With improved analytical methods, and with greater awareness of the potential health danger, many studies have been carried out in recent years at much lower blood levels, often in new directions (e.g. cellular respiration and ion transport). There has been a rush to establish whether there are any ill-effects due to the lower blood levels typical of the inhabitants of many modern cities and their suburbs. Disquieting observations of the diverse effects of lead at relatively low concentrations have been described.

Moreover, the hematopoietic system, which is involved in so many bodily functions, is clearly affected at very low blood lead levels. The disturbances of certain enzymatic processes involved in heme biosynthesis are of particular concern. Heme is an essential constituent of hemoglobin, the respiratory pigment of the red cells (erythrocytes) through which the blood transports oxygen to the tissues, as well as several related hemoproteins, such as the cytochromes. These compounds control cellular energetics and some metabolize toxic substances in the liver.

As cells are exposed to increasing concentrations of lead, impairment of many other tissues becomes progressively more evident. Some of the effects on metabolic functions at the cellular and subcellular levels and on the nervous system, the cardiovascular system (with particular reference to blood pressure), reproduction and growth will now be discussed. Table VII.I shows the blood lead levels at which the onset of these effects becomes apparent.

(a) Heme Biosynthesis

A simplified diagram of heme biosynthesis indicating the points at which lead affects the process is presented in Figure VII.1. Many of the reactions concerned take place in the mitochondria of kidney, liver and brain cells. Lead interferes with certain key reactions in two ways, either inhibiting some

Table VII.I

Blood Lead Concentrations at which 10 and 50% of Subjects Exhibit Effects above the Upper Limit of Normal*

	Blood lead concentrations (µg/dL)			
	Adults		Children	
Effect	10%	50%	10%	50%
ALA dehydratase	9 ± 2	18 ± 3	11 ± 2	20 ± 2
EP	19 ± 3	33 ± 5	21 ± 3	36 ± 4
Urine ALA	34 ± 4	58 ± 5	38 ± 19	66 ± 47
Urine coproporphyrin	37 ± 5	58 ± 9	41 ± 17	74 ± 24
Anemia	63 ± 11	120 ± 52	25 ± 8	54 ± 13

* Values given represent mean ± 2 SEM.

Source: Piotrowski and O'Brien, 1980

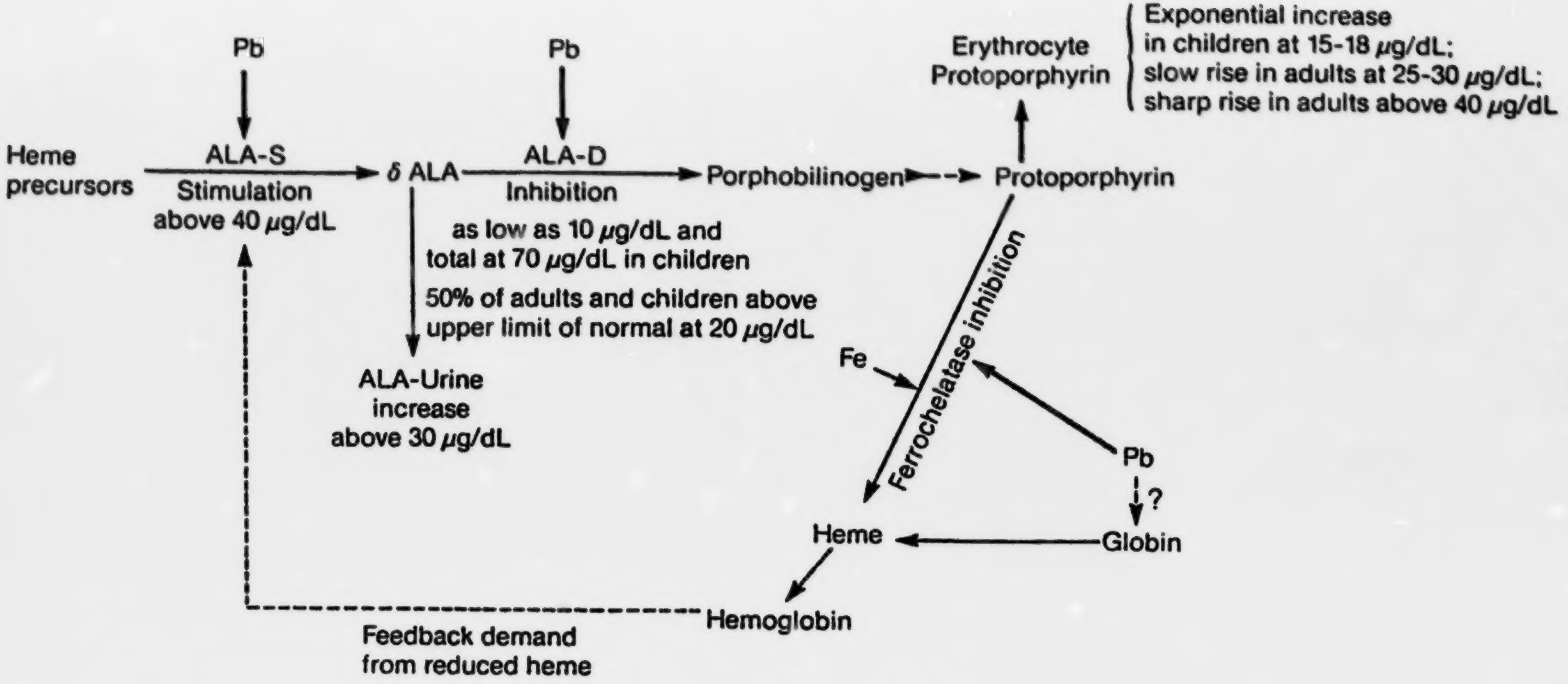
Pb
Pb
Heme precursors
ALA-S
Stimulation above 40 μg/dL
δ ALA
ALA-D
Inhibition
as low as 10 μg/dL and total at 70 μg/dL in children
50% of adults and children above upper limit of normal at 20 μg/dL
ALA-Urine increase above 30 μg/dL
Porphobilinogen
Protoporphyrin
Erythrocyte Protoporphyrin
Exponential increase in children at 15-18 μg/dL; slow rise in adults at 25-30 μg/dL; sharp rise in adults above 40 μg/dL
Ferrochelatase inhibition
Fe
Pb
?
Globin
Heme
Hemoglobin
Feedback demand from reduced heme

Figure VII.1. Biosynthesis of heme – a schematic.

Source: This report.

directly, or stimulating others to produce unusable quantities of the neurotoxic intermediate compound, δ-aminolevulinic acid (ALA).

Four separate but related mechanisms of lead interference result in accumulation of ALA in the body fluids:

- lead directly stimulates the enzyme ALA-synthetase (ALA-S) to produce ALA from heme precursors

- lead inhibits the activity of ALA-dehydratase (ALA-D), which produces porphobilinogen by condensation of two molecules of ALA

- lead inhibits the enzyme ferrochelatase, preventing the insertion of iron into the porphyrin ring in the final step of the process. This allows the entry of zinc into the ring, forming the very stable--and biologically inactive--compound zinc protoporphyrin, which endures for the entire life of the cell (120 d)

- the reduced production of heme exerts feedback pressure on the first step of the process, stimulating the formation of yet more ALA

Inhibition of ALA-D activity occurs at blood lead levels as low as 10 μg/dL (Tola et al., 1973), and it is virtually total at about 70 μg/dL; however, it does not appear to cause a decrease in heme biosynthesis until ALA-D levels have fallen to 20-30% of normal (Hernberg, 1980). Piotrowski and O'Brien (1980), however, showed that about half of all adults and children show effects typically above the upper limit of normal at blood lead levels of 18-20 μg/dL, and 10% do so at levels as low as 9 μg/dL. Urinary ALA increases markedly above 30 μg/dL blood lead. This indicates excretion of accumulated ALA, probably mostly derived from ALA-D blockage as ALA-S stimulation is believed to occur only at blood lead levels in excess of 40 μg/dL. ALA-D activity is affected mainly in the red blood cells, but Secchi et al. (1974) noted that it occurs in liver cells. In children, ALA-D activity is probably also reduced in brain cells (Millar et al., 1970).

Also of importance is the behaviour of erythrocyte protoporphyrin (EP). Piomelli et al. (1982) observed an exponential increase in EP above an apparent blood lead threshold of 15-18 μg/dL in 2004 children, 1852 of whom had blood lead levels below 30 μg/dL. Fully half the children had significant elevations of EP at a blood level of 30 μg/dL. For adults, a slow rise in EP seems to start at about 25-30 μg/dL (Roels et al., 1976), and a dramatic increase occurs at about 40 μg/dL (Moore and Goldberg, 1985). Urinary coproporphyrin, also originating from ferrochelatase inhibition, starts to increase at blood lead levels of 35-40 μg/dL.

Controversy remains as to whether these effects represent adverse health impacts or harm. Indeed, Moore and Goldberg (1985) asserted that "None of these effects can be classified as biologically important until the degree of impairment is sufficiently large to be reflected in a decrement in the synthesis of the final product, i.e., hemoglobin" (and other hemoproteins such as cytochrome P-450).

The wide range of individual and racial tolerances that occur in populations makes it clearly impossible to ascribe clinically harmful effects to the lowest blood lead levels mentioned above. It is equally impossible, however, to deny that the changes in the biochemical processes reported reflect a marked potential change in the ability of the system to cope with the lead insult. These changes appear well established at blood lead levels very close to those typical of the Canadian population. Indeed, Piotrowski and O'Brien (1980) show the onset of anemia in 10% of children at blood lead levels as low as 25 µg/dL.

(b) Interference with the Formation of Hemoglobin

The biosynthesis of globin, the protein moiety of hemoglobin, appears to be inhibited as a result of lead exposure (Dresner et al., 1982), the effects being mediated through a disturbance in heme biosynthesis. A deficiency of hemoglobin and a shortened erythrocyte survival due to direct cell damage appear at a blood lead threshold of about 40 µg/dL in children and 50 µg/dL in adults. A reduced uptake of iron has often been found in both adults and children exposed to high concentrations of lead. The resultant anemia may be severe.

(c) Heme Biosynthesis and Nerve Tissue

Three possibilities are suggested by animal studies:

- ALA, of which there is an excess, is neurotoxic. *In vitro* studies suggest that this is so (Silbergeld and Lamon, 1980)
- heme deficiencies in tissues external to the nervous system, notably the liver, may result in damage to nerve tissue. It has been observed that decreased levels of heme in the liver result in elevations of tryptophan, serotonin and 5-hydroxy indoleacetic acid in the brain, where they may cause damage. These increases are reversed by infusions of heme (Litman and Correia, 1983)

- there is a reduction of heme in neural tissue, which renders the tissue prone to injury from lead (Whetsell and Kappas, 1981; Whetsell et al., 1984)

(d) Lead and Mitochondria

There is an extensive body of evidence pointing to lead as a cause of structural and functional damage to the mitochondria. Silbergeld et al. (1980) showed that lead enters the brain mitochondria. They isolated rat cortical endothelial cells and exposed them to lead *in vitro*. Using electron microscopy and X-ray microprobe analysis, they demonstrated electron-dense lead deposits within the mitochondria. They also observed that lead appeared to be accumulated in the same mitochondrial areas as calcium. Their results suggested that "lead is preferentially sequestered in mitochondria of capillary endothelial cells. Further this selective localization may be associated with lead-induced disruptions in intracellular calcium metabolism and trans-epithelial transport processes."

This suggestion received strong support from the work of Pounds et al. (1982a, b), among others, whose studies confirmed the easy entry of lead into cells and cellular compartments and its disturbance of intracellular ion transport, particularly in neural cell mitochondria where there is a high capability for calcium transport.

In most studies, *structural* changes in the mitochondria occurred only at relatively high concentrations of lead, but Fowler et al. (1980) observed swollen mitochondria in rats chronically exposed to lead from gestation to 9 months of age at a dietary lead dosing level as low as 50 μg/g and a median blood lead level of 26 μg/dL (range 15-41 μg/dL).

Many _functional_ changes in the mitochondria attributed to lead have been described, including the following:

- ferrochelatase inhibition. Actually, many other enzymes involved in the formation of heme are found, if not formed, in the mitochondria of all organs in which heme synthesis takes place (brain, kidney, liver, spleen, heart and lungs), and lead interferes with the formation and activity of several of these
- oxidative phosphorylation. This important mitochondrial function is also affected by lead. According to Silbergeld (1984), "_In vivo_ exposure is reported to cause reductions or delays in the developmental increases in concentrations of cerebral cytochromes C and C1, and also changes in the ADP-independent respiration. Some of these effects may be due to heme synthesis inhibition, resulting in decreases in the availability of heme for cytochromes. However, the ability of lead to alter mitochondrial respiration _in vitro_ in cerebral tissues from unexposed rats suggests that some aspects of this toxicity do not depend upon decreased cytochrome content"
- cellular calcium metabolism. The presence of lead in the mitochondria of brain capillary endothelial cells is associated with an increased accumulation of calcium, and will cause apparent changes of calcium efflux from the mitochondria (Silbergeld, 1984). Similar demonstrations of the effect of lead on mitochondrial calcium metabolism have been made using rat hepatocytes (Pounds et al., 1982a, b). Rosen (HWR) summarized the position:

> "....it may be concluded that lead ion per se modifies transcellular uptake and release of calcium, i.e. lead produces an increase in cellular uptake of calcium and inhibits its efflux. It appears, therefore, that this pervasive toxic effect of lead at low concentration occurs in tissues of diverse function: transepithelial transport processes of calcium are diffusely impaired. It is reasonable to suggest that this toxic effect of lead on normal cellular transport of calcium is one common mechanism by which lead interferes with the normal functioning of several different cell types. This is not meant to be an exclusive concept."

The low concentrations mentioned above refer, in most cases, to the concentration of lead in a culture medium or a fluid in which a tissue is bathed. For instance:

- the observation (Pounds et al., 1982a, b) that the addition of lead to the culture medium at concentrations as low as 10 μM markedly increased the uptake of calcium by hepatocytes
- the observation of Silbergeld et al. (1980) that capillaries suspended in a solution containing 0.1 mM lead nitrate showed irregularly shaped granules inside the mitochondria

Important as they obviously are, these findings cannot readily be related to *in vivo* blood lead levels.

(e) Lead and the Vitamin D Endocrine System

Rosen et al. (1980) found that children with blood lead concentrations between 33 and 120 μg/dL have markedly reduced serum values of a metabolite of vitamin D, 1,25-dihydroxy vitamin D (1,25-$(OH)_2$D). Following chelation, the blood concentrations of both lead and 1,25-$(OH)_2$D returned to normal within a short period. These workers concluded that the lead ion impairs the metabolism of vitamin D, though the possibility that 1,25-$(OH)_2D$ was converted by lead into an inactive product could not be ruled out.

This compound is involved in bone mineralization, the intestinal absorption of phosphorus and the absorption and homeostasis of calcium, an essential element in a number of neurological, pancreatic and other response mechanisms (Rosen et al., 1983). 1,25-$(OH)_2D$ is produced in the mitochondria of kidney cells by a process that involves a hemoprotein, cytochrome. It is depressed at blood lead concentrations of about 33 µg/dL, the level at which erythrocyte protoporphyrin accumulation becomes noticeable (Rosen et al., 1980, 1981). The involvement of the hemoprotein and the appearance of erythrocyte protoporphyrin as its action is inhibited might suggest a lead-originated interference with heme production.

Decreased calcium, depression of bone mineralization and increased levels of parathyroid hormone have been observed at blood lead levels in excess of 62 µg/dL. There have been suggestions of effects below 20 µg/dL, but DeLuca (1985) stated it was premature to conclude that lead specifically suppresses the vitamin D hormone system.

However, in view of the multiple functions of vitamin D, it is possible that interference by lead with its metabolism may lead indirectly to widespread deleterious effects.

5. Neurobehavioural Effects of Lead Exposure

Lead appears to be selectively absorbed by cells of the central nervous system, especially during its early development. As a neurotoxin, it appears to affect sodium and calcium metabolism and to inhibit the neuronal enzymes. Accumulation of the heme precursor ALA is also believed to be linked to some of the clinical and biochemical effects of lead intoxication. Acute encephalopathy (degenerative brain disease) occurs in adults at 100-120 µg/dL (Chisholm, 1965; NAS, 1972), and in the most susceptible children at 80-100 µg/dL (EPA, 1977).

Much of the controversy surrounding the effects of lead exposure, especially in young children, has arisen from research suggesting that significant neurobehavioural and neurophysiological effects occur at blood lead levels typical of those widely found in urban populations. These suggestions have created alarm among parents and neighbourhood groups, and have led to strong political pressure for action to reduce lead exposure still further.

The controversy as to which specific effects are related to particular levels of exposure to lead remains unresolved. Most critical is the question of whether there is a direct association between lead and neurobehavioural effects, or whether it is poverty that leads to neurobehavioural problems that in turn tend to increase lead exposure. Or, even more simply, do deprived conditions increase both lead exposure and neurobehavioural deficits?

Obviously, the question of what comes first can be answered only through investigational designs that are prospective in nature. In the early cross-sectional and retrospective studies, it appeared that blood lead levels in the order of 50-70 µg/dL were associated with an average decrease of about 5 points in child IQ (de la Burde and Choate, 1972, 1975; Rummo, 1974; Rummo et al., 1979; Rutter, 1980). Most of the above studies have, however, been criticized (Bornschein, HWR; Dietrich, HWR; Yule and Rutter, 1985; EPA, 1986), the most relevant criticism being that the studies had in general failed to make sufficient allowance for a number of factors, mostly related to the socio-economic situations known to influence child IQ's.

Needleman et al. (1979) correlated neurobehavioural deficits with dentine lead. They found an effect in a large population of Boston children at dentine levels subsequently shown to correspond to blood lead levels in excess of 33 µg/dL. Though initially criticized, a significant part of this group's work has now been generally accepted (Winneke, 1983; Needleman, 1984; Needleman et al., 1985; Yule and Rutter, 1985).

Yule and Rutter (1985) undertook an extensive review and reanalysis of a number of neuropsychological investigations of children published since 1979, in an effort to discern whether there is a consistent pattern of results across studies. Children in these studies had blood lead levels ranging from 0.5 to 35.5 µg/dL, and dentine or whole tooth lead concentrations ranged from 1.4 to 38.5 µg/g. Following detailed consideration of technical and methodological aspects of this body of research, the authors concluded that "the neuropsychological effects of lead within the range seen today in ordinary environments without special high risks are quite small". This conclusion takes account of both statistically significant and statistically nonsignificant studies. The authors judged that lead accounts for not more than 2-3% of the variance of cognitive performance in children. This is equivalent to the 2- to 5-IQ-points difference often observed between low- and high-lead groups in the studies referred to. In population terms, however, a 5-point shift in IQ means a doubling of children with IQ less than 70 (Rutter, 1980; Needleman, 1983; HWR). All these studies with positive findings report differences in the same direction.

The arguments just outlined are very important, though they do not answer the crucial point of a cause-effect relationship: the full interplay of the triad socio-economic status-lead-IQ still remains to be elucidated.

However, the results are at least consistent with a causal hypothesis, because (i) they are biologically plausible, (ii) statistical control of confounding variables has not completely removed the effect of lead, and (iii) there is agreement between studies as to the direction of the results and, to a degree, the magnitude of the effect (Yule and Rutter, 1985). As to the preliminary results from prospective studies, Bornschein (HWR) reported on five of them; there appears to be an early measurable effect (a lowering of the Bayley Motor Development Index [MDI] at 6 months of age and a decreased birth weight) attributable to intra-uterine exposure to lead. This effect may not be persistent, and no associations have been found with post-natal blood lead levels. A pertinent observation was made by Zeskind and Ramey (1981), who demonstrated the overwhelming effect of infant stimulation on IQ during the first 3 years of life: the detrimental effect from foetal malnutrition became progressively worse in a 36-month follow-up of infants reared in a non-supportive environment (cognitive general index [CGI] 71 ± 10); the high-risk infants who received additional stimulation had a CGI of 96 ± 11, indistinguishable from that of the low-risk group receiving comparable stimulation (CGI 98 ± 16).

Another important point made by Yule and Rutter (1985) and by Dietrich (HWR) is that the association between lead and lowered IQ appears to operate only within socially disadvantaged children. Two more points are relevant:

- there is no clear evidence that the body burden of lead in children induces hyperactivity. Yule and Rutter (1985) believed that "it would be premature to rule out the possibility of an effect, but, equally, there are no strong grounds at present for inferring one"

- uncertainty remains with respect to a threshold for neuropsychological effects of lead. Negative results have been observed in studies in which the mean blood lead concentrations are well below 15 μg/dL, such as British work reviewed by Harvey (1984). By contrast, the US studies, which provide the strongest evidence for such effects, involve populations with substantially higher blood lead levels. Yule and Rutter (1985) considered that "it is possible (although not demonstrated) that lead effects are negligible with blood levels below 20 μg/dL"

6. Other Effects on the Central Nervous System

A few studies have attempted to explore the relationship between blood lead levels and electrophysiological functions in children. These studies were prompted by findings of marked electroencephalographic abnormalities in lead-intoxicated animals (Zook et al., 1972; Saito, 1973). Alteration of visual activity, decreased scotopic visual acuity and alteration of conduction velocities in visual pathways have been reported by Fox et al. (1982), Impelman et al. (1982) and Cooper et al. (1980). Alterations in visual evoked responses (VER) to light flashes were also reported by Fox et al. (1977, 1979) and Winneke (1979). However, the findings--alterations in response, latencies or amplitudes--had inconsistent patterns that Otto (1986) thought were probably due to differences in methodology, though it was possible that a real effect existed as regards brainstem auditory evoked responses. The studies conducted on children at lead levels below 60 μg/dL show equally inconsistent patterns of results and lack confirmation in replication studies (Otto, 1986). The biological significance of the results reported is as yet unknown, and their relationship to psychological functioning is not yet fully understood. These studies should still be considered experimental.

7. Effects on the Peripheral Nervous System

More precise methodology and clearer, more interpretable results are available for assessing the effects of blood lead levels and changes on the peripheral nervous system. The well-known symptom of wrist drop in lead-intoxicated workers spurred investigations on the nerve conduction of lead workers. The earlier studies were conducted on individuals whose lead levels approached toxicity (greater than 80 μg/dL), whereas later studies sought to clarify the effects at lower lead exposures (blood lead levels less than 70 μg/dL). The Commission has reviewed a large number of studies conducted in these ranges of lead levels.

Several difficulties mar the interpretation of the results (Neri and Bichlmeier-Callery, HWR). Some of the studies are weak in their control of extraneous and confounding variables, some in the choice of controls and some in the methodology used in measurements. No consistency has been found as to which of the several measurements used and which of the different nerves tested represent the most sensitive indicator for the effect sought. Differences in the methodologies used by the various investigators may have contributed to this problem. It is clear, however, that at or near toxic levels there can be severe damage to the peripheral nerves. The pathological changes are mostly axonal degeneration with loss of the large myelinated fibres, accompanied by some demyelination (Simpson et al., 1964; Behse et al., 1972; Buchtal and Behse, 1979). This contrasts with the findings on animals, where the primary lesion seems to be segmental demyelination (Fullerton, 1966). The diameter of the nerve fibres (axons) and the degree of myelination are important factors, in that conduction velocity in the nerves increases with both diameter and myelination of the nerve fibres. The types of changes described as a consequence of lead toxicity also occur in the case of most other chemical toxicants. It is, therefore, very important to have control of these exposures (alcohol, other heavy metals, etc.) in order to sort out their effects from that of lead.

Most studies have found alterations in one or more of the measures of nerve conduction used. Though no one measure is clearly more sensitive to lead insult than others, the most frequent finding is a reduction in the conduction velocity of the sensory fibres of the median nerve (Seppalainan et al., 1975; Araki and Honma, 1976; Ashby, 1980).

The same uncertainty prevails below toxic levels as for other health effects attributed to lead. Most investigators report findings at blood lead levels above 60 µg/dL. Only four studies report effects at levels lower than 50 µg/dL. These include the two Seppalainen studies (Seppalainen et al., 1979; Seppalainen and Hernberg, 1982) in which there are some uncertainties as to the assessment of exposure. In the Seppalainen and Hernberg (1982) study, most of the significant differences were found in the range 50-59 µg/dL. The third study is by Bordo et al. (1982), who showed that impairment was not found in the workers with the longest exposure in the group whose blood levels were never above 50 µg/dL. Fourth is the study by Rosen et al. (1983) which used current blood lead levels of workers in the lower exposure group (less than 30 µg/dL) who had employment histories ranging from 18 to 336 months. It is likely that this level had been substantially exceeded in the past. One of the better studies, a matched-pair analysis by Triebig et al. (1984), found significant differences on nerve conduction only at levels above 70 µg/dL.

Though several investigators have reported negative findings, there is little doubt that nerve conduction is altered at levels as low as 50-60 µg/dL. No evidence has been found that such effects can occur at lower levels. The clinical importance of the changes at lead levels below 70 µg/dL is not known. There are at least some indications (Bordo et al., 1982) that these changes may spontaneously reverse themselves.

8. Effects of Lead on Reproduction

That lead at toxic levels is capable of affecting fertility in men, as well as reducing the viability of the conceptus (miscarriage and self-induced abortions), has been known from the mid-19th century (Paul, 1860; Neri, HWR). Several studies have shown that lead (and other heavy metals) crosses the placental barrier (Angell and Lavery, 1982; Barltrop, 1969); in addition, during pregnancy there is frequently a mobilization of calcium (and consequently of lead) stores from the bone because of increased calcium requirements (Thompson and Robertson, 1985). These factors and the concurrent high prevalence of anemia (with its inherent enhancement by lead toxicity) make pregnant women and their foetuses a high-risk group for toxic effects from lead. Lead is also found in the milk of lactating women, but it is unlikely that the very small concentrations involved adversely affect the breast-fed infant (Rockway et al., 1984; Kovar et al., 1984; Rabinowitz et al., 1985).

9. Prenatal Exposure

Maternal lead poisoning may cause infant encephalopathy and other neurological defects (Pearl and Boxt, 1980; Sensirivatana et al., 1983). However, the literature indicates that many of these effects may be reversible (Neri, HWR; Qazi et al., 1980; Pearl and Boxt, 1980). Several epidemiological studies have been conducted since the mid-1970's to clarify the relationship between lead exposure and spontaneous abortion (Wibberley et al., 1977; Nordstrom et al., 1978; Alexander and Delves, 1981). For most of them, the exposure to lead was assumed on the basis of the residential histories of the parents. The results from these studies are not conclusive. Furthermore, because the studies are ecological in nature, the effect of other environmental (other heavy metals) variables, life-style (cigarette smoking, alcohol) and social factors (institutional status, work situation) could not be taken into account (Neri, HWR). Similarly, no clear relationship has been established for an effect of lead in sudden infant death syndrome (Hoppenbrouwers et al., 1981; Erickson et al., 1983; Lyngbye et al., 1985).

10. Male Fertility

The toxic effect of lead on male fertility appears to be more clearly established, particularly at elevated blood lead levels (greater than 60-70 µg/dL) (Lancranjan et al., 1975; Butrimovitz et al., 1983; Dawson et al., 1985). At such exposure levels, lead has been found to affect spermatogenesis, potency and libido; specific pathological changes have been demonstrated by testicular biopsies (Braunstein et al., 1978). Most studies have been conducted on lead-exposed workers. Though some of the results appear to be contradictory, there is little doubt that lead was responsible for the changes reported. However, these changes are not specific to lead, as cadmium, copper, zinc and other metals frequently handled by lead workers have also been implicated (Stanwell-Smith et al., 1983; Dawson et al., 1985). As well, coexistent urogenital conditions and the effect of life-style variables (drinking, smoking) have rarely been controlled. Effects of lead on male fertility have not been demonstrated at the levels to which the general population is exposed.

11. Mutagenic Effects

There have been many studies on the possible mutagenic effects of lead. During the last 15 years, the majority have concentrated on occupationally exposed individuals. The type of mutagenic effects reported by the various investigators are mostly of the so-called minor type, consisting of chromosome breaks and gaps and chromatid exchanges (Lehnert, 1970). In those cases in which other metals were also measured, the association with lead was found to be less strong than with other metals (Neri, HWR; Deknudt et al., 1973). Most positive results were reported at the higher blood lead levels (above about 50 µg/dL). Several studies show that these changes can be reversible. Again, in most cases, personal habits such as smoking are well-known inducers of chromatid aberrations (Neri, HWR; Nordenson et al., 1978a, b). Ionizing radiation exposure, particularly medical X-ray examinations routinely conducted in workers, is also a well-known inducer of chromosomal aberrations, and was not taken into account. The relationship between lead exposure and mutagenic effects on humans therefore remains uncertain.

Evidence from animal experiments and human studies leaves little doubt that exposure to high levels of lead has important reproductive effects. It is very doubtful, however, whether such effects occur at lower exposures, such as those of the majority of the Canadian population (less than 20 μg/dL). The evidence at such lower dosages becomes less consistent, both in human observations and animal experiments, and the effect of possible confounders such as exposure to other toxic metals, life-style habits and socio-economic situations increases substantially. The Commission feels that well-designed epidemiological studies involving a large number of subjects are needed to clarify the relationship.

12. Lead and Blood Pressure

The relationship between blood pressure and blood lead has been a focus of controversy ever since an association between the two was found in an analysis of NHANES II data (Harlan et al., 1985; Pirkle et al., 1985; Schwartz, HWR). Controversy was also aroused by the use of a time trend analysis (i) to support a claim that reduction of lead in gasoline was responsible for the declining trend of blood pressure in US white males aged 40-59, and (ii) to make an estimate of the saving in lives from reduction of complications due to high blood pressure. Harlan et al. (1985) clearly stated that "No causal inferences should be drawn from this cross-sectional survey about the blood pressure effects of lead, calcium, and zinc, although these observations from the general population are consistent with extensive animal studies of lead toxic effects and trace metal interactions," and that "Our analyses of the NHANES II data provide a link between blood lead levels and blood pressure, but the cross-sectional nature of the survey limits inferences of causality." The Commission agrees with these statements.

The Commission has reviewed in detail the literature on the association between blood lead and blood pressure. The conclusion drawn from the review is that, though the association is biologically plausible, there is not enough evidence for assurance of a cause-effect relationship from the findings reported by the various investigators. The major problems in the interpretation of the NHANES II analysis are (i) the failure to present data on blood pressure elevations in a more realistic or categorical way, such as cross-tabulations and relative risks (cross-tabulations would more readily permit the reader to assess the details of the association); (ii) the almost exclusive reliance on statistical probability as a measure of the strength of the association, rather than on the measurement of the effect (regression coefficient) and the proportion of variance explained by the regression (R^2); and (iii) the focus in the subsequent workup of the data of only one age-sex-race group, which was done to exclude potential confounding variables.

The Commission has examined the hypothesis as regards Canadian conditions. Neri and Hewitt (1986) have analysed the data from the Canada Health Survey 1978-1979, the only similar survey in North America, and two industrial cohorts. One cohort comprised 115 employees of Cominco Ltd., for which up to 5 yearly determinations of concomitant blood pressure and lead levels were available. The second cohort was a group of 534 workers at Brunswick Mining and Smelting Corporation, for whom up to 13 years of concomitant measurements were available. This last data set also includes 6 years of measurements of urinary cadmium.

Data from the Canada Health Survey (which have the advantage of having been collected over a period of 9 months, in contrast to 49 months for the NHANES II data), provide weak qualitative support for the results reported by

Harlan et al. (1985) and Pirkle et al. (1985); both female and male changes are in the direction expected by the hypothesis of a lead effect. However, the association is weak. Its significance disappears when hemoglobin levels and copper (both of which were used in the NHANES analysis) are fed into the regression. More importantly, the relationship is weak because the effect of raising blood lead levels from 1 to 43 µg/dL is only in the order of 1.5 to at most 3 mm Hg blood pressure. The proportion of blood pressure variance explained is also quite small. The analysis of one cohort provided no support for the lead hypothesis. Data from the other cohort, in which blood pressure measurements were judged more reliable, did provide statistically significant support for the hypothesis in relation to diastolic, but not systolic, blood pressure.

The Commission concludes that the association between blood lead levels and blood pressure in white males aged 40-59 in the NHANES II survey (Schwartz, HWR) is established, but remains to be demonstrated as a phenomenon typical of all groups. As to whether the association is causal, we are still uncertain. The unadjusted correlation coefficients reported by Harlan et al. (1985) show that the proportion of variance in blood pressure explained by lead is 2.5% for white and 1.2% for black males. In the Canada Health Survey, the corresponding value is 1.4% for both sexes.

In light of the weakness of the effect noted, even if the association is found to be causal, its importance from the point of view of prevention of cardiovascular disease may not be as substantial as implied by Pirkle et al. (1985). We do not believe that useful estimates of health costs in Canada can be based on these correlations.

13. What Blood Lead Levels Prevail in Canada?

The foregoing account makes clear the importance of blood lead levels as the usual measure of human exposure to lead, and as indicators of the likelihood of adverse health effects. It is generally agreed that blood lead levels are the best available measure for such purposes, though in children particularly they are far from ideal. What is the characteristic range of blood lead levels in Canada, and how do these compare with levels in other countries?

A detailed review of the available evidence is given in HWR by Neri and Tessier, and of levels of lead exposure by Nantel. A summary of US evidence is given by Schwartz. Here we comment on these detailed analyses, and attempt to generalize the results.

The Commission was surprised at the paucity of epidemiological surveys of blood lead conducted on a national scale in Canada and elsewhere. This lack of hard data precludes categorical statements about our relative situations. Only two population surveys have been conducted with the exclusive aim of assessing blood lead levels: the Ontario survey (Ontario Ministry of Health, 1985), exclusively on children, and a series of surveys by the Commission of the European Communities (1981), involving both children and adults. Blood lead level was included among the measurements taken in two other major population surveys, the United States NHANES II (1970-1976) (Mahaffey et al., 1982; Annest, 1983) and the 1979-1980 Canada Health Survey (1981). However, there are substantial reservations as to the validity of both surveys in the estimation of blood lead levels in their "mother" populations. NHANES II was conducted over a period of 49 months, during which time substantial changes occurred in the blood lead levels of the population sampled. The survey was not designed to assess such unsuspected changes, and therefore the results obtained are more suggestive than representative of a shift in population blood lead levels.

The lead estimates derived from the Canada Health Survey have also been the subject of several criticisms. These have been discussed in detail in HWR; however, the whole, the Commission is inclined to agree that the estimates reported can be used for comparison purposes, provided that excessive emphasis is not placed on the exactness of the mean levels reported, particularly in the lower ranges (< 5 µg/dL).

Two methods of comparison are possible for most of the countries for which data have been reviewed. One is to look at the way in which levels in the various age-sex groups sampled from the general population compare with Canadian data, and the second is to examine the levels in those groups living in known high-exposure areas (hot spots), and for which more studies have been reported.

Two measures can be used in comparing different groups: the mean levels for the group, and the proportion of individuals within the group who are above a given level. The second is preferable, for a number of reasons. Arithmetic means are not really sufficient to describe a distribution of values without a measure of their dispersion, such as the spread, or range, and the standard deviation. In addition, as with most biological values, the distribution of blood lead levels in populations is skewed to the right. The median and the geometric mean are, therefore, better descriptive parameters. However, investigators do not all use the same parameters, and comparison becomes difficult. This problem may largely be overcome by using the proportion of individuals above certain values, even though the values chosen may not always be the same. This approach has the additional advantage of being less dependent on the accuracy of measurements that are less precise at lower lead levels.

A number of random (or quasi-random) sample surveys of blood lead levels published in the last decade for non-occupational groups of populations, particularly children, have been reviewed. These consist of larger and smaller populations, populations in high-exposure areas and urban and rural populations. Most surveys have focussed on special groups, either because they were known to be located in high-exposure areas, or because they were special-risk groups (such as children). Understandably, large-scale population surveys can be implemented only by government agencies. Blood lead levels appear to have decreased in most industrialized countries. The Commission believes that it would be of great use to keep track of these trends, but we also feel that the levels ascertained in the latest Canadian surveys are reassuring enough that the large costs necessary for a dedicated survey of this type are not justified. However, we recommend that lead measurements be taken whenever large surveys are conducted for other purposes. In such cases it is imperative that a rigorous methodology of sample collection and chemical analysis be followed, so as to avoid the controversy that has marred the lead measurements of the Canada Health Survey. Blood lead surveys should also be a prime mode of assessment for the hot spot problem.

There are significant discrepancies between the values for blood lead in children obtained from capillary blood (fingerpricks) and others obtained from venous blood. In particular, we were impressed by the low levels of lead reported in a recent survey of children living in central London, UK (Strehlow and Barltrop, 1986). These levels were similar to those reported in the Canada Health Survey. The Commission agrees with the investigators that the most likely explanation for the difference in lead levels between the two types of blood sampling is external contamination*.

The implication of these findings is the need to take into account the methods used in blood collection when results from various surveys involving children are compared. The Commission strongly recommends that when surveys of blood lead are conducted, whether in adult or child populations, strict, well-standardized and control routines for blood collection and storage be adopted. Venous blood only should be used (for children and adults alike), unless experienced persons who have been specifically trained for pinprick blood collection are available.

The results of the comparison of blood lead levels between Canada and those countries for which comparable data were obtained were reassuring (Figures VII.2 to VII.4). Canadians seem to have more favourable levels of blood lead than the populations of some other industrialized countries of Europe (see Figures VII.3 and VII.4). Also of relevance are the data in children reported by McCabe (HWR), indicating the large proportion of American children found with elevated (greater than 40 μg/dL) blood lead levels, and his important note on lead-containing paint. Rosen (HWR) stated that at his hospital alone, 120-150 children are admitted annually for chelation therapy. This compares with an average for the past 5 years of eight admissions in all Canada (see Table I.I, Section I, and Appendix VI). There is no doubt, however, that blood lead levels in the US have fallen since the data shown in Figures VII.3 and 4. In the last quarter (1980) of the NHANES II analysis, mean US population blood lead levels had fallen to 9.8 μg/dL (Grant, 1986).

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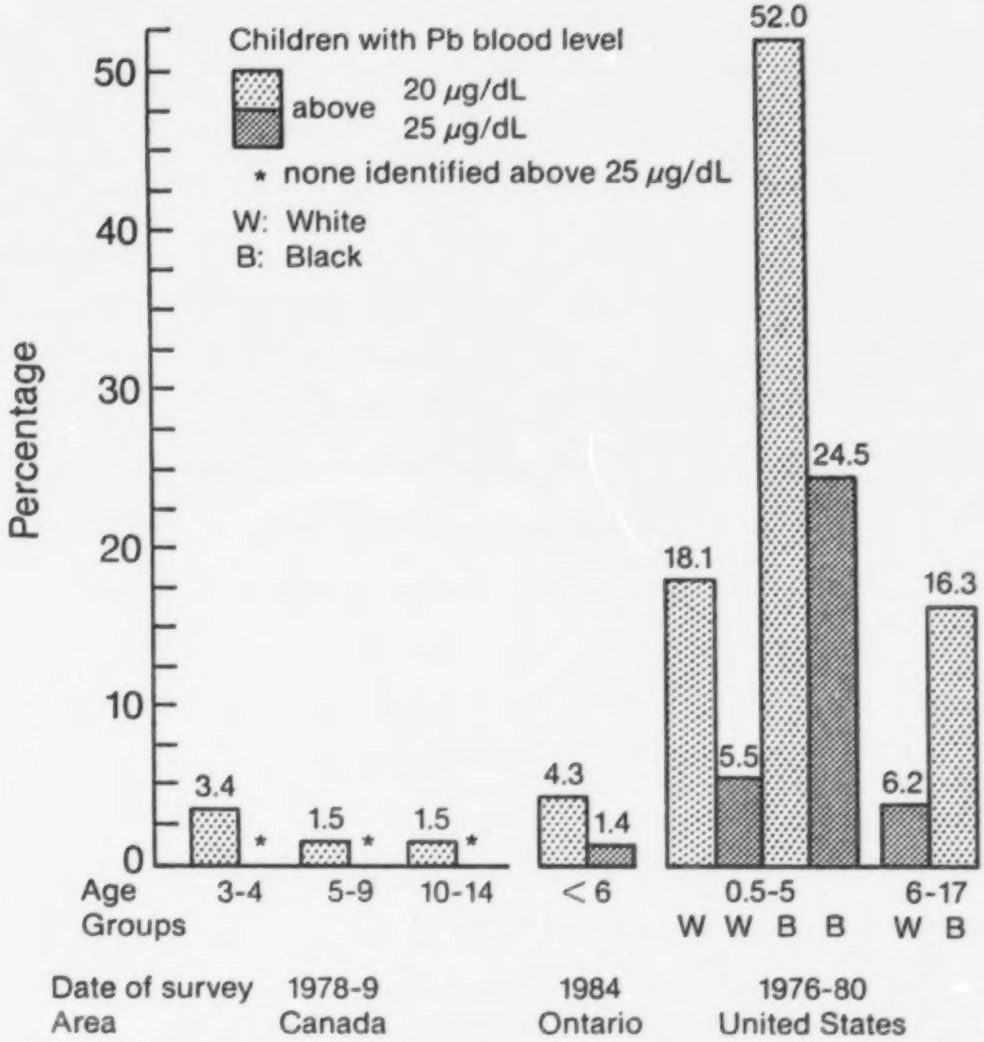
* A group of 108 children aged 6 months to 5 years examined in NHANES II by pinprick methods had mean blood lead levels no less than 6 μg/dL higher than children sampled by venipuncture (Mahaffey et al., 1982).

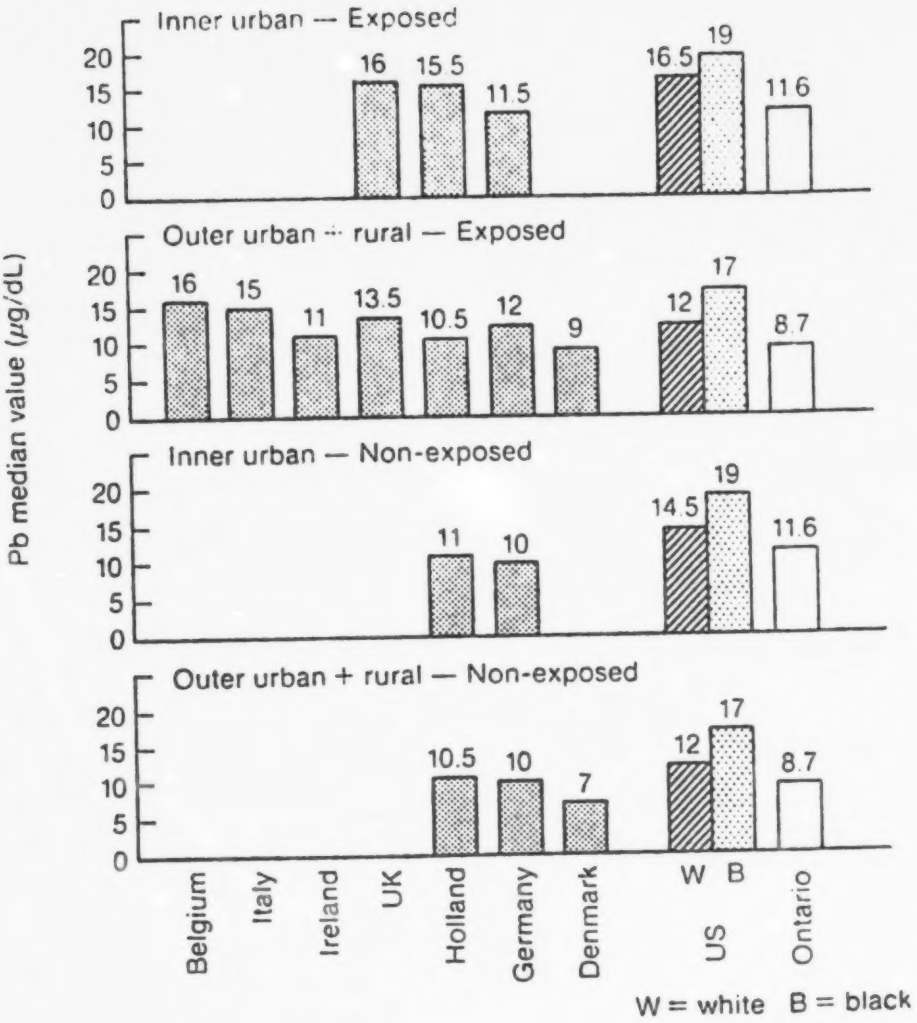


N.B. Blood leads have been falling in many, if not all the countries shown in Figures VII.2 and VII.3. Data from different time periods should only be compared with caution.

Figures above columns are actual percentages.

Figure VII.2. Comparison of children with blood lead levels above 20 and 25 μg/dL for Canada, Ontario and United States.

Source: Canada Health Survey (1981). Ontario Ministry of Health (1985). Annest (1983).



N.B. Ontario data are not separated between exposed and non-exposed
Canada Health Survey (Unseparated) median was 7 µg/dL

. Blood leads have been falling in many, if not all the countries shown in Figures VII.2 and VII.3. Data from different time periods should only be compared with caution.

Figures above columns are actual percentages

Figure VII.3. Comparison of median blood lead values (µg/dL) among children of specified age groups in the following areas:

Sources: United States NHANES II (US), 6 mos - 5 yrs, 1970-76 (W-white, B-black)
Selected European Community countries, under 14 years, 1977
Canada Health Survey, 3-9 yrs, 1978-79
Ontario Blood Lead Survey, under 6 yrs, 1984.

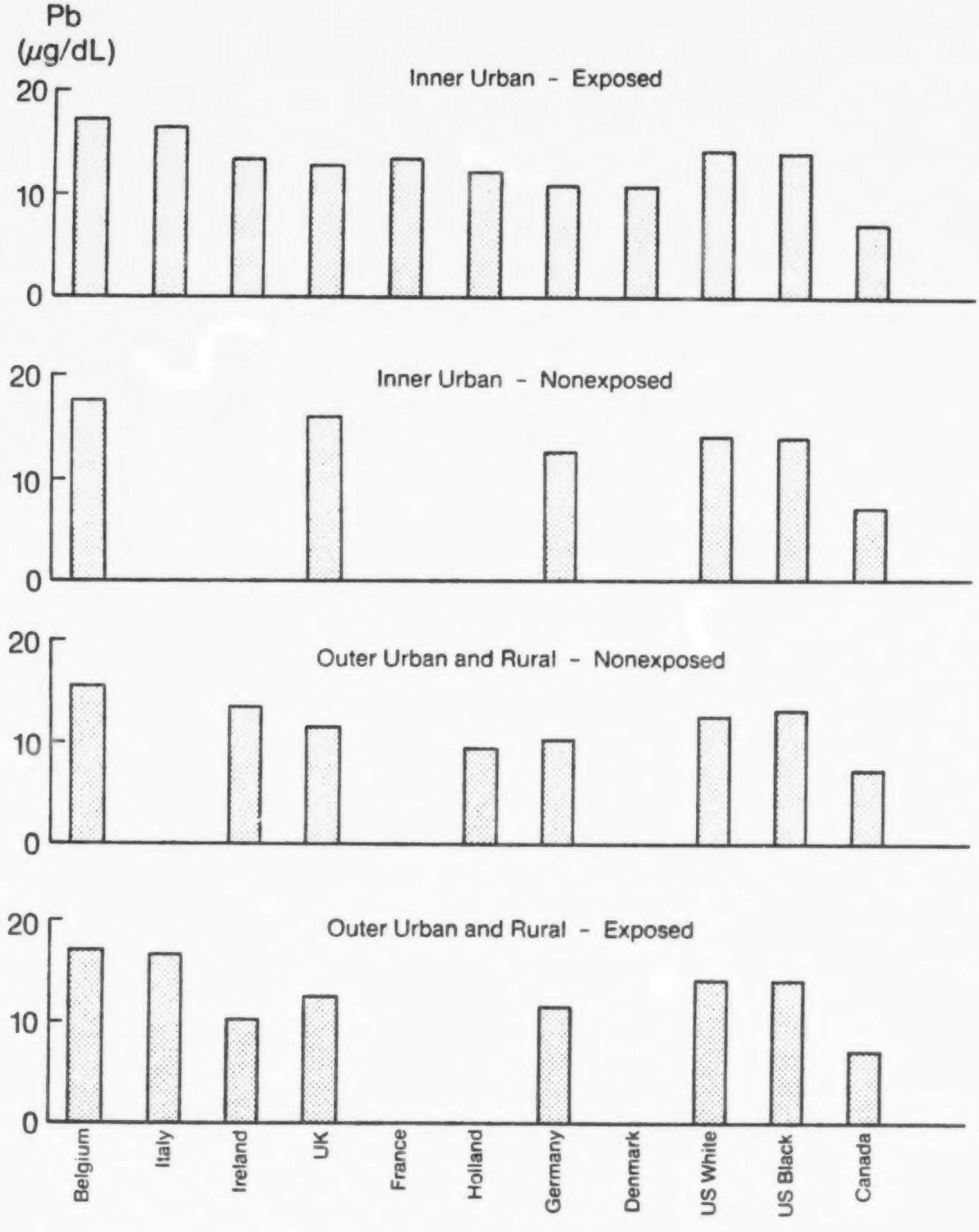


Figure VII.4. Comparison of specified average blood lead values among adults separated by location in the following areas:
United States NHANES II (US), 1976-80, geometric means
Selected European Community Countries, 1977, medians
Canada Health Survey, 1978-79, arithmetic means

Sources: CEC, 1981
Annest, 1983
Canada Health Survey, 1981.

Undoubtedly, the past regulation of lead content in Canada in many materials has been responsible for much of the continued improvement. Canada has also been fortunate in that it does not have large areas of deteriorating houses. Both factors are considered of great importance in the US where, according to Pope (1986), estimates of up to 842 000 children under the age of 7 may be living in houses with lead paint in deteriorating condition (EPA, 1986). Even in the so-called hot spots in Canada, such as Trail and South Riverdale, lead levels in children (and adults) are not as high as in similar areas of the US, and the proportion of children with blood lead concentrations above the action level (25 µg/dL) is substantially less than in the cores of the large urbanized areas of other countries (Table VII.II).

14. What is the Overall Health Effect?

We have seen that blood lead levels in Canada, to the extent that we can rely on the sparse data available, seem to be lower than in other industrialized countries, and there is some evidence that they have been falling; we decided, therefore, to confine our attention to the possibility that significant health effects may occur at blood lead levels below the 25 µg/dL level so often cited as an intervention level.

There is no doubt that the hematopoietic system--the system including the synthesis in the body of heme and its successors and the role of blood in other bodily functions--is disturbed at these low levels. Heme synthesis itself is altered down to almost the lowest levels observable. It was these facts that led the Commission to agree in its interim report that there was a good case for reducing blood lead levels below 1985 levels. The case is still good in 1986. It is not clear that actual harm results from this disturbance,

Table VII.II

Mean Blood Lead Levels Found in Various Surveys of Hot Spots* in Canada and Other Countries

Locality	Year	Mean blood lead level (µg/dL)	Age group	No.
Trail, BC (all within 2 miles)	1977	22.0	1-3 and 6	147
		28.0		49
South Riverdale (Toronto), Ont.	1973	28.0	?	
	1982	14.0	6	135
	1983	15.8		70
	1984	15.5		216
Rouyn-Noranda, PQ	1979	25.0	0-12	
Murdochville, PQ	1983	16.0	6-12	327
Silver Valley (USA)	1977			
Area 1**		65.6	1-9	
Area 2		47.6		
Area 3		33.8		
Area 4		32.3		
Belgium smelter area***	1974	30.1	11	37
	1975	26.4	11	40
	1976	24.6	11	38
	1977	28.9	11	56
	1978	27.8	11	43

* Areas around smelter.
** According to distance from smelter.
*** Locality not identified, 1 km from smelter.

Source: Summarized from Neri and Tessier, HWR but, as we saw, harm is hard to define. As Southwood (1983) stated, the case for reducing blood lead levels rests on prudence, not certainty of harm.

But what of the claims that have been made that there are threats to behaviour and mental development in children, to neuropsychological performance and even to growth and bodily development? What of the blood pressure issue?

It has emerged that there are grounds for supposing that lead has real neurobehavioural effects on children. The effects shown by the tests are, however, small, and tend to be swamped by confounding factors related in particular to socio-economic status. Moreover, it is hard to detect these effects at the blood lead levels observed in Canadian children. Other effects on children are also hard to demonstrate at such levels. The same is even more true of suggested effects on growth and development.

Much the same is true of the lead and blood pressure relationship. There is a weak association between blood lead levels and blood pressure, at least among certain groups of adults. But this, too, is hard to disentangle from confounding factors. At the low blood lead levels typical of Canada's population, we doubt whether this relationship has much, if any, importance.

Do these uncertainties mean that Canada can ignore the lead issue? Most certainly not. There are still hot-spot areas in which fairly high blood lead levels occur among children. These are discussed in Section VIII. There is also the matter of occupational exposures, which lie outside our terms of reference. Blood lead levels among the exposed work-force are much higher than in the general population. The relevance of recent research to their health and prospects is high. We hope that others will examine it.

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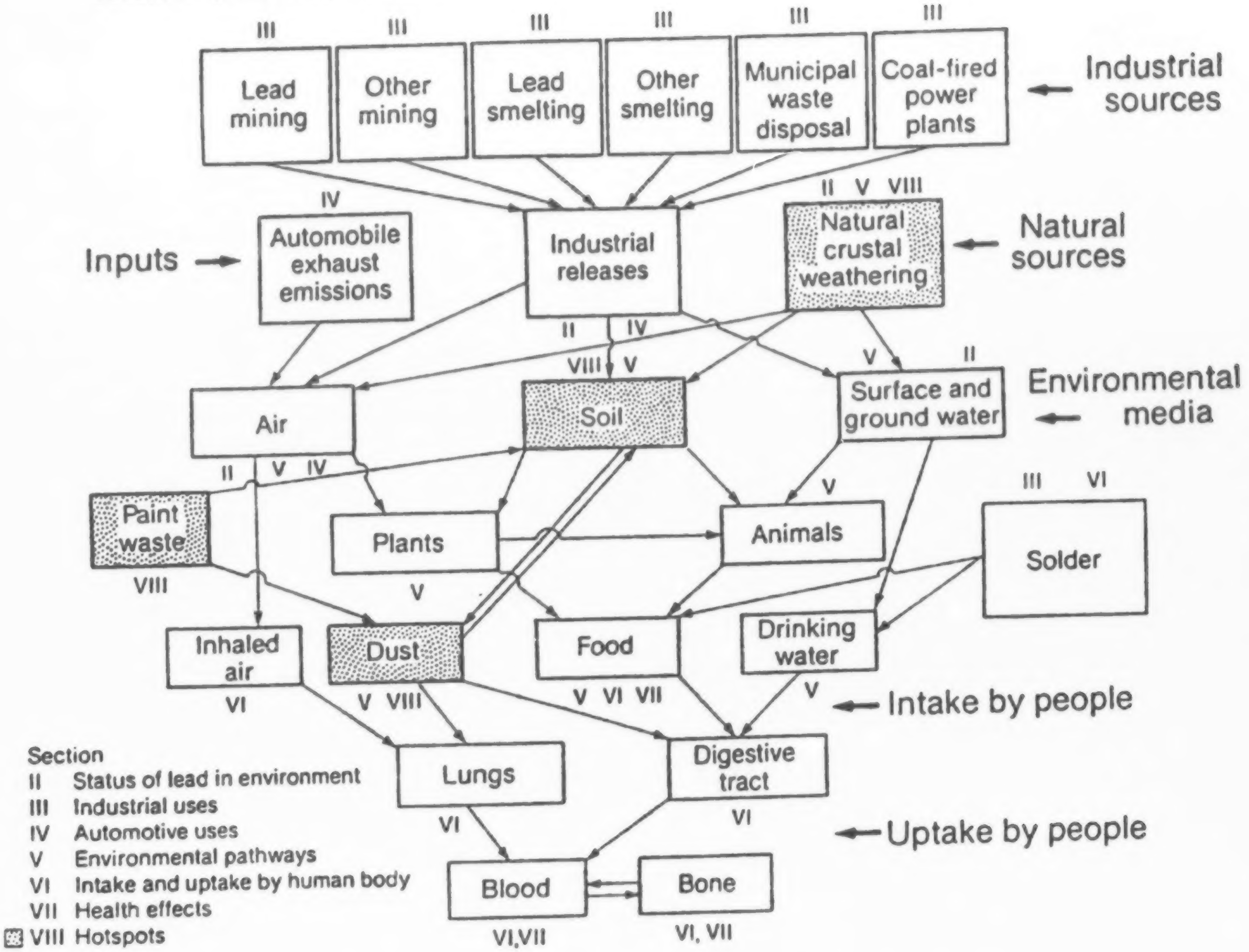

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Lead mining
III
Other mining
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III
Other smelting
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▨ VIII Hotspots

Area covered in Section VIII

VIII The Problem of Hot Spots

1. What are Hot Spots?

We have sketched a picture of Canada as a country where lead is widely dispersed through the environment, especially near where people live. We have suggested that most Canadians are still less exposed to lead than are the populations of other industrialized countries; they appear to have lower body burdens, and to be less at risk from lead as an environmental toxin.

This optimistic view is not justified for areas where concentrations of lead in air, water or soil are sharply higher because of local industrial sources. These areas are usually called hot spots. Public perception of the hazards posed by such hot spots has given this issue political visibility. Hot spots are of two main kinds:

- downtown areas near lead smelters or lead-using industries, where industrial overspills are supplemented by lead from gasoline as a result of heavy traffic

- areas near primary lead smelters, mines or concentrators, as well as other metallurgical industries; these areas are usually remote from large cities, but include service towns and villages where exposures may be high

In this section we have grouped together cases of both kinds that have caused concern. The treatment is deliberately anecdotal, in an effort to convey a sense of the real, local experiences that lie behind the generalities of the previous sections. We have included a brief review of a characteristic urban overspill--lead-contaminated sewage sludge, often used as a fertilizer on agricultural land.

In hot spots, unusually high levels of lead occur in soil, plants and surface waters (not always all three). Ambient air lead concentrations are also usually increased, as is lead in dust-fall. In cases where the industry responsible for the problem has ceased operation or curtailed emissions, the contamination of soil and surface water may persist for decades or even centuries, though ambient air concentration will usually be only slightly raised (from local dust pick-up).

We noted above that lead is persistent, once deposited in soil, because of its chemical immobility. Hence, contaminated sites remain a hazard long after they have been abandoned. Much of the lead in soils and sediments is in fact a legacy from the past, even where operations still continue. Cleaning it up poses real legal, economic and political problems.

We have been unable to prepare a complete list of hot spots in Canada. Some remain unidentified. We discussed the primary lead smelters, mines and refineries in Section III. Of these, Kimberley and Trail, British Columbia, and Belledune-Bathurst, New Brunswick, were identified to the Commission by public interest groups as being of concern on environmental grounds. We visited these areas, with the exception of Kimberley. Actually, iron and steel plants and copper and nickel refineries may be among the largest emitters of lead (Fenco, 1985), and may create hot-spot conditions. However, they do not appear to have attracted public attention as lead emitters (see Section III), and there is a general lack of information about their impact.

Because of the depressed state of the mining and smelting industries, we were not surprised to receive, from local interest groups, pleas that we not add further to the environmental control costs of primary industry's installations. Citizens' groups and union leaders joined with local authority officials and company officers in making such pleas.

It was from well-organized and determined neighbourhood groups in large cities that the Commission received its strongest representations. These groups are concerned with conditions in residential areas surrounding secondary lead smelters. There is no doubt that these areas are the homes of those Canadians most obviously at risk, members of the lead-working labour force excepted.

The Commission suspects that in other Canadian localities there are smaller hot spots that have escaped detection. One such case, in Kingston, Ontario, was investigated recently (Stokes and McConnell, 1984). We believe that measures should be taken to identify such areas of unsuspected contamination, where the original source of the lead has been forgotten or overlooked. Old industrial records, anecdotal evidence, geochemical surveys and the work of urban geographers are among the resources that might be tapped in starting such a survey. It has also been suggested that the native peoples of British Columbia and New Brunswick may have roasted galena (lead sulphide) to produce lead oxide pigments. Local high concentrations may hence be found near former villages.

2. Urban Hot Spots: Secondary Lead Smelters

Secondary lead smelters have become important to the functioning of the modern lead industry (Section III). They take scrap lead in various forms, particularly batteries, and recycle the metal and other useful products to a variety of users. Because the industry depends heavily on local sources of supply, it always seeks to locate near or within large urban areas. The surviving smelters in Canada are in the Vancouver district, Winnipeg, Greater Toronto, Mississauga and Greater Montreal. Others have existed, but are now closed down. Figure III.3 (see Section III) shows the installations still active.

The very nature of the secondary lead smelting business implies an operation difficult to control for environmental overspill. Battery crushing, for example, is a necessary first step in recycling the small mountain of old car batteries that become redundant every day. Plastic, acid, lead and lead oxides are released, and their overspills have to be contained. Much of this is done by small operators whose methods are primitive, and usually uncontrolled. But the major perceived problems that have arisen have been located near larger, integrated operations working from raw scrap to finished lead--ingots, castings, various compounds and alloys. We shall look at two particular cases, both in Toronto.

(a) South Riverdale, Toronto*

South Riverdale is an old residential district in eastern downtown Toronto. It lies east of the Don Valley (and hence the Don Valley Parkway), south of Queen Street, west of Woodbine Avenue and north of the Toronto Terminals Railway tracks (the eastern approaches to Union Station). The Gardiner Expressway also flanks the neighbourhood on the south. The residential areas south of Queen Street East are largely occupied by small houses, whose population is ethnically and socially diverse, and is mostly drawn from the lower income groups. Along and south of Eastern Avenue, this community faces downtown Toronto's major surviving industrial area.

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* We are indebted to the South Riverdale Community Health Centre and to Canada Metal Company, especially Mr. P.J. Irwin, for help in preparing this section.

Conspicuous among the plants involved is that of Canada Metal Company Ltd., whose Eastern Avenue plant, established in 1925, is one of Canada's largest secondary lead smelters. It smelts and refines lead from scrap materials produced elsewhere (no battery crushing is carried on at the plant). It also produces lead oxides in a parallel assembly. Figures III.5 and III.6 (Section III) illustrate the two operations. One other lead-using plant was located nearby, a paint and lead chromate manufacturing facility that is now defunct. It must have contributed to the local soil burden.

The local community has been apprehensive for 20 years about the effects of lead overspills. The South Riverdale Community Health Centre is the present-day focus of community efforts. The Commission contracted for an analysis of the local situation from this Centre. Their responses are reprinted in full in Report VI, which outlines the sources of concern and recommends further action. This citizens' group has been among the most active in the country in hot-spot issues.

Children in the area have exhibited above normal-values of blood lead since an initial survey in 1973. This survey showed that 13% of South Riverdale children aged under 6 years had blood lead levels above 40 μg/dL (a figure comparable with conditions in the most affected areas of US cities). It was assumed that these levels among children came primarily from ingestion of dust or soil transferred to fingers, food and candy. Emissions from Canada Metal and from leaded gasoline were seen as the local sources. It was feared, but not proven, that flaking lead paint might also be a factor. Soil lead levels were very high near the plant, with an exponential decrease with distance from the source (Roberts et al., 1974).

The Ontario Ministry of the Environment issued a control order on the plant in 1974. Emissions remained high until 1979, and then fell to more acceptable levels by 1981. They have remained fairly constant to the present time*. The improvement has been due to the installation of modern ventilating equipment and baghouses, improved processing, curtaining of apertures, improved handling of scrap and waste materials and cleaning of outside surfaces (to remove dust that might become fugitive). Contact between company officials and neighbourhood groups seems effective.

In an analysis for the Commission (on behalf of the South Riverdale Community Health Centre), Rachlis and Hertzman (1986) concluded as follows:

- Children aged under 6 years living near the plant (within about 300 m, the area with soil lead levels above 1000 µg/g (ppm) had in 1984 blood lead levels averaging 15.5 µg/dL, compared with a value of 12.0 µg/dL in Toronto and other Ontario urban sites. 18.5% of South Riverdale children tested had blood lead levels above 20 µg/dL, compared with 5.6% for other Ontario urban sites. Living near the plant thus still imposed a substantial penalty on children, notwithstanding the greatly improved environmental performance. Air lead levels in the neighbourhood were similar to those elsewhere in Toronto (0.3-0.5 $\mu g/m^3$). The authors concluded that the causal factor was the uptake of soil lead, accumulated in the soil over a long period of time. The Commission agrees that soil is likely to be an important factor in this instance, but believes that current and recent fallout are the key dietary source.

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* The 1974 levels laid down by the Ontario Ministry of the Environment were a maximum 24-h criterion of 5 $\mu g/m^3$ and a 30-d maximum of 2 $\mu g/m^3$. Canada Metal, in its brief to the Commission, spoke of only one exceedance of the 5 $\mu g/m^3$ 24-h criterion in the past 4 years. Inspection of the records confirms general compliance with the criterion since 1981, though there was one exceedance of the 24-h criterion in April, 1986 (to 7.1 $\mu g/m^3$).

- These figures represent a sharp improvement over 1973, when mean blood lead levels were 28 µg/dL, with 13% of cases above 40 µg/dL. Since 1982, however, no further improvement has been observed.

- Blood lead levels in children, given variations between individuals, need to be lowered substantially, probably to a mean value of 8.5 µg/dL, which the authors believed would ensure that 99% of all children would have levels below 20 µg/dL.

- This should be accomplished primarily by removal of soils with lead contents above 500 µg/g (soils had already been removed from properties with lead contents above 2600 µg/g, the provincial guideline proposed in 1974).

- Action to reduce gasoline lead emissions is still important if this removal is to be effective.

- Because peeling paint has been observed in some houses where children have blood levels above 20 µg/dL, inspection of such premises should be initiated.

The improvement in South Riverdale, which is encouraging yet incomplete, can be ascribed to strong community action; to alert responses by the public health authorities; to sound technical measures by the corporation concerned; to useful monitoring programmes of air quality and blood lead levels; to public information programmes; and to good scientific back-up from provincial government sources and university and private laboratories. There has also been much improved co-operative action between the provincial Ministry of the Environment and the local health authorities. These improvements will still be crucial if environmental quality is to be raised to the levels typical of other urban areas. Many of those concerned spoke to us of their frustrations at the slowness of progress, and the effort involved in getting co-ordinated action by the many participants. This was certainly true a decade ago. It is much less the case in 1986. There is wide agreement on the need for further action, and some recognition that the several participants have to "get their act together".

(b) The Niagara (Toronto) Neighbourhood*

This Toronto neighourhood lies in a comparable situation to South Riverdale; it stands just west of downtown between King Street West and the Toronto Terminals Railway, with the Gardiner Expressway paralleling the latter, and lying even closer to the small residential area. Bathurst Street lies on the east, and Shaw Street on the west. A belt of industrial properties, mostly old, extends between Niagara and Wellington Streets and the railway (whose own extensive yards, now being reduced, also flank the neighbourhood).

The area has very heavy traffic, and hence has collected substantial lead-containing dust of gasoline origin. But a main lead source is the secondary lead smelter operated by Toronto Refiners and Smelters, Ltd., a small family-owned business in the south-eastern corner of the neighbourhood, on Bathurst Street. This plant has operated since 1945 (incorporated in 1944), and is a fully integrated smelter. Output includes lead ingots and alloys. Its raw material is largely old car batteries (a high proportion coming from the US). The plant has a large battery-crushing machine, whose capacity exceeds local sources of supply.

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* We are indebted to the Niagara Neighbourhood Association and to Toronto Refiners and Smelters Ltd. for help in preparing the section.

Concern arose in the early 1970's about noise, emissions and fugitive dust from the plant. Lead was seen as accumulating in the soils. Soil lead levels were very high near the plant, decreasing exponentially with distance (Roberts et al., 1974). Some of the most affected properties have since undergone soil removal. In 1972-1974, blood lead levels in the local population were high. 3.1% of the 734 persons tested had levels of 40 µg/dL or over. 9.5% were above 30 µg/dL. Strong pressure was brought by the Niagara Neighbourhood Association and other groups for improvements in environmental controls. The company was prosecuted under the Environmental Protection Act in 1977, and there followed a series of appeals that resulted in the company's acquittal. Relations between neighbourhood groups, the company and municipal and provincial authorities were tense.

Today things are improved. Many families with children have left the neighbourhood, which is in the process of redevelopment. Blood lead levels in children under the age of 6 in 1984 averaged 12.4 µg/dL, and, of the few whose level was over 20 µg/dL, subsequent re-examination (of venous blood) showed no case with confirmed levels above this point. Measures taken by the company have reduced particulate lead emissions. The Association, however, complains of broken windows and broken curtains around the active areas of the plant, and reports exceedances of air quality criteria. One such exceedance occurred in April, 1986, but 30-d criteria were easily met. The Association also calls for full enclosure of the furnace building complex. We understand that the company is undertaking an agreed programme of improvements, in search of an Ontario Ministry of the Environment programme approval. Nevertheless, the Association is pressing matters through the courts. Like its neighbours in South Riverdale, it argues that soil removal within the affected areas is the key to a further drop in blood lead levels. The reduction in use of leaded gasoline has, of course, helped reduce the dust burden, and will continue to help in the next few years. But the Association asserts, and the Commission agrees, that the soil will continue to be an undesirable source of lead for years to come, though we stress that current and recent dust-fall contribute most to dietary uptake. The Association also argues strongly for education of residents as a means of accelerating the improvement.

The Toronto Refiners and Smelters plant raises the question of decommissioning. A proposal is being examined for the westward extension of Front Street. This would require the demolition of the plant, and of several neighbouring properties. Given the large amounts of lead that impregnate the buildings, floors, foundations and surrounding yards, this operation (if it goes forward) will require extensive soil and waste removal, and replacement with other materials low in toxic substances.

Meanwhile, the company is left in doubt about its own future, at a time of low profitability in the industry as a whole. As its President pointed out to us, finding a new site would be very difficult; he intends to continue operating on the present site as long as he can. There is little incentive for the company to carry out local site improvements under these conditions of uncertainty.

3. Urban Hot Spots: Metalworking Industries*

Many other kinds of metalworking industries are distributed throughout the urban areas of Canada. Some of these have become lead hot spots, even though lead-working may not be the main objective. Because the primary iron and steel industry may emit not much less lead than do the primary lead smelters (see Table III.III, Section III), it is reasonable to ask whether lead pollution may not affect the surroundings of other metalworking plants where lead is used as an input, or is released as an unwanted by-product. The Commission has no complete list of such sites, but has examined cases where lead pollution has been alleged to exist.

The City of Winnipeg has two secondary lead smelters, and a foundry which works more generally in the alloys field, chiefly to supply aviation parts. All three are in the western and north-western outskirts of the city, but only one (Canadian Bronze Company Ltd.) has residential areas nearby. In 1979, the Province of Manitoba began soil testing in the vicinity, and found elevated lead levels at some sites. Concern arose that elevated lead levels might occur in the yards of neighourhood schools. Many residents suspected that the operations of Canadian Bronze were responsible.

In 1982, a comprehensive survey of lead levels was conducted in the area, and an air sampling programme was carried out to identify the characteristic speciation or fingerprinting of lead in the area. The work was led by Canadian Bronze, in collaboration with Atomic Energy of Canada Ltd. at Pinawa, under contract to the Ministry of the Environment. The research showed that the particulate lead collected had a signature (i.e. size distribution and chemical species) typical of automobile exhausts, rather than of smelter emissions. Emissions from Canadian Bronze were thus not a major factor. The report on this study (Wotton and Doern, 1983) is a small classic of environmental science, and deserves to be imitated elsewhere.

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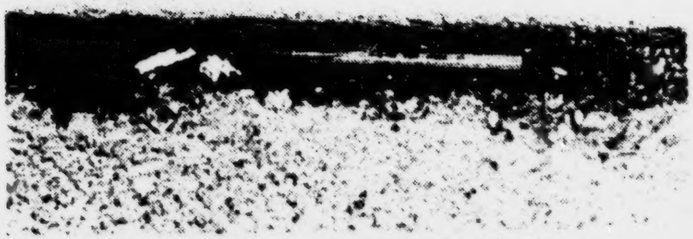
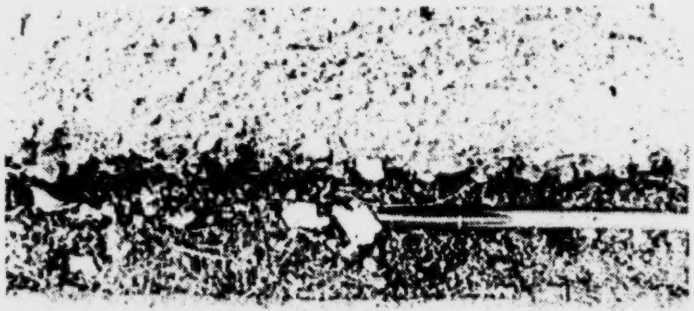
* We are grateful to Dr. Tom Owen, Deputy Minister of the Environment, Government of Manitoba, and his colleagues of the Environmental Management Division, for much help in preparing this section.

Soil surveys were conducted in the vicinity of the plant. Lead levels were at or above 2600 μg/g (the then Ontario guideline, in the absence of Manitoba guidelines) on 31 private properties (including gardens), on a picnic site maintained by Canadian Bronze and on a number of grass verges and boulevard medians of city streets. Inspection of the dusts cleansed from the yard of the Weston School nearby showed high levels of lead. An initial removal of dust from the yard in 1981 had shown levels in the range of 4000-5000 μg/g. A year after the clean-up the lead levels had returned to their unsatisfactory condition, in the range 950-6000 μg/g. Contaminated soil was removed from most of these sites in the summer of 1982, and the school yard was refurbished.

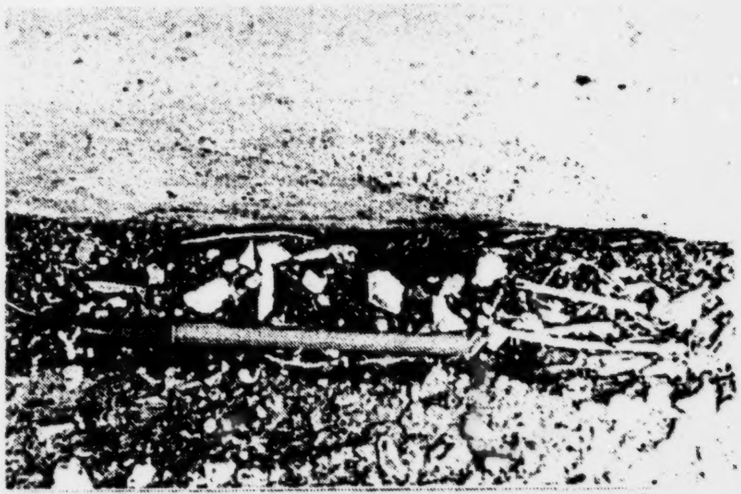
Subsequent study of other school yards in Winnipeg showed some uncomfortably high levels, but nothing approaching the Weston problem. Detailed studies showed that lead in dust was highest near busy streets (Jones and Wotton, 1984). Flaking exterior leaded paint was a contributor in Weston (see Figure VIII.1), this being one of the few Canadian cases to come to the Commission's attention where paint has been definitely identified as a significant source of lead in dust.



Paint chips at base of building
48 hours post clean-up



Putty found at base of building
48 hours post clean-up



Prior to clean-up old paint flakes ↑
↓ and putty are readily found in
accumulated soil and flakes

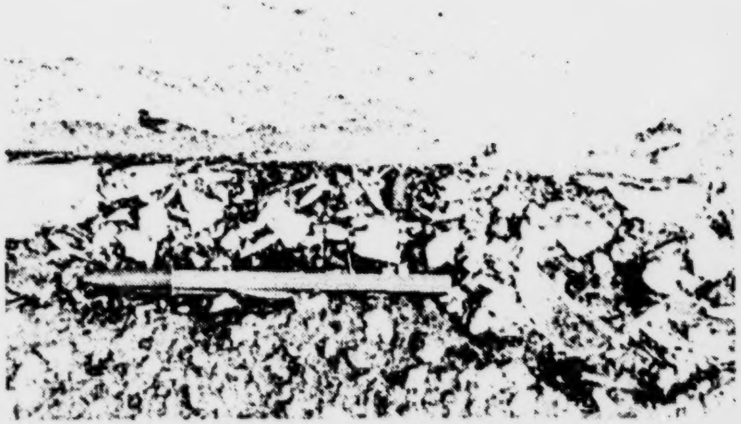


Figure VIII.1. Paint flaking; Weston School, Winnipeg.

Source: Manitoba Environmental Workplace Safety and Health Department, 1986.

The Weston School experience differs from the Toronto cases in a number of ways. Community pressure seems to have played a smaller role in Winnipeg. Though the issue arose because of fears of overspill from a lead-using plant, detailed research identified leaded gasoline as the main source of the pollution. The proportion of leaded gasoline in total sales is larger in Winnipeg than in Toronto.

Dust and soil are a prime source of lead for children playing out of doors and probably for gardeners, though the lack of blood lead data at Weston prevents certainty as to the local effect. Soil removal was practised at all three sites, but only at levels exceeding the Ontario guideline of 2600 µg/g. The Commission supports this practice, but believes that the old Ontario guideline is too high (see pp. 269-271). Clearly, the time to clean up such soils will be immediately following the drop in gasoline lead expected in late 1987, i.e. in the summer of 1988. Otherwise, as Weston School showed, a new deposition of lead will occur on soils and sealed surfaces. It is also essential to make sure that the replacement soil is low in lead, and that adequate disposal sites for the contaminated materials are available.

4. Primary Lead Industry: Environmental Problems

The large scale of Canadian primary mining and smelting operations (see Section III) suggested that problems of lead exposure might occur in their vicinity. No complaints reached the Commission after we had advertised our concerns. Accordingly, we approached the operators of the largest facilities, as well as local authorities, unions and public interest groups. We organized public meetings in or near the localities, and visited the production facilities. Two such studies were completed:

- Cominco Ltd.'s facilities at Trail, BC (smelting and refining), and Kimberley, BC (mining) (not visited), together the country's largest lead-zinc producers
- Brunswick Mining and Smelting Corporation's facilities at Belledune, NB (lead smelting and refining; sulphuric acid and fertilizer production), Bathurst and Dalhousie, NB (mining, concentrating and shipping)

In both locations, all concerned persons and institutions collaborated fully with the Commission, and spoke almost with one mind about the present situation--that there had been major problems in the past; that the responsible company had responded with control and clean-up measures that had satisfied local and provincial authorities; that adequate local collaboration now existed between the actors to monitor the environmental situation; and, above all, that all parties feared for the future of the lead industry, and therefore their jobs. To quote an unusually frank brief to the Commission from Local 7085 of the United Steelworkers of America in Belledune, NB:

> "... the reduction in the lead market has already had harsh effects on an area already suffering from excessive high unemployment and we are greatly concerned that further pressure on lead markets will compound this already unacceptable situation.

And the more emphatic words of the Mayor of Jacquet River, NB, a village that is home to many lead workers:

> "... a discontinuation of lead production in our area would condemn our village. Many of our young people would be forced to leave. For those remaining the future would be very bleak. In essence all the vitality would be sapped from our community....We are proud of the Village of Jacquet River and are very sensitive to the needs of its citizens. In the past we have not felt threatened by the exposure to lead and have benefitted from its production during the past twenty years. We surely hope to benefit from its future production."

Similar views were expressed to us by many individuals in both areas visited. The fear of becoming ghost towns is a recurrent nightmare in Canadian mining and smelting communities.

(a) The Belledune-Bathurst-Dalhousie Area, NB*

Complex lead-zinc sulphide ores in prodigious quantities underlie the forested hills of northern New Brunswick. Three mining operations have been developed: Brunswick 12 (in full operation), Brunswick 6 and the Heath-Steele Mine (the last two are both currently closed). These are located south of Bathurst, NB. At Brunswick 12, the ore is concentrated and separated from other components, chiefly zinc sulphide and several other metallic compounds. The lead concentrate is railed to Belledune, NB, 30 km north of Bathurst, directly on the south shore of the Baie des Chaleurs. At Belledune it is smelted and refined. The by-product sulphuric acid is used as input to a chemical fertilizer plant, within the same corporate fence. All these facilities, from mine to fertilizer plant, are the property of Brunswick Mining and Smelting Corporation Ltd., which is controlled by Noranda Inc. Shipping facilities for concentrates are at Dalhousie, NB.

The mining operation involves primarily inorganic lead (insoluble sulphides). Fugitive emissions to the atmosphere from the mining and concentrating operations at Brunswick 12 in 1985 have been estimated to be 180 t/a (New Brunswick Department of Municipal Affairs and Environment, 1986).

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* We are indebted to many people for information on the facilities in this area. In particular, the Department of Municipal Affairs and Environment prepared an excellent brief, as did the Belledune Environmental Monitoring Committee (Catherine MacLaggan, Chairman). Brunswick Mining and Smelting Corporation went to much trouble to let us see what we wanted to see. We also appreciated the help of the Bedford Institute of Oceanography.

Effluents to local surface waters have been reduced from 33 t in 1982 to 1.2 t in 1985. The area is very thickly forested with second-growth trees, and most large particulate emissions are probably deposited within this forest. Effluents, however, tend to accumulate in stream sediments or marine deposits near the discharge point. Earlier sterilization of the Little River by acid mine and tailings drainage has now been corrected, and fish have reappeared.

At Belledune, emissions from the smelter complex amounted to 59 t of lead per year in 1984, but rose to 90 t in 1985, because of problems with the baghouse operation (New Brunswick Department of Municipal Affairs and Environment, 1986) (see Figure VIII.2). They have since fallen significantly. Such particulate matter is chemically more active than waste from mining and milling. Effluents from the complex went into Belledune Harbour until 1980, but are now discharged in the open bay. Lead discharges since 1981 have been in the range 0.2-0.3 t/a.

Prior to 1981, heavy metals effluent from the plant constituted a major problem. Belledune Harbour's sediments are heavily contaminated with cadmium and zinc (Hildebrand, 1984; New Brunswick Department of Municipal Affairs and Environment, 1986). The smelter's effluents were clearly implicated. Accordingly, an advanced treatment system was installed, and discharge was removed from the harbour to more open waters. Table VIII.I shows the excellent result. Recent monitoring indicates that concentrations of heavy metals in harbour sediments have been greatly reduced*, as have concentrations of metals in solution.

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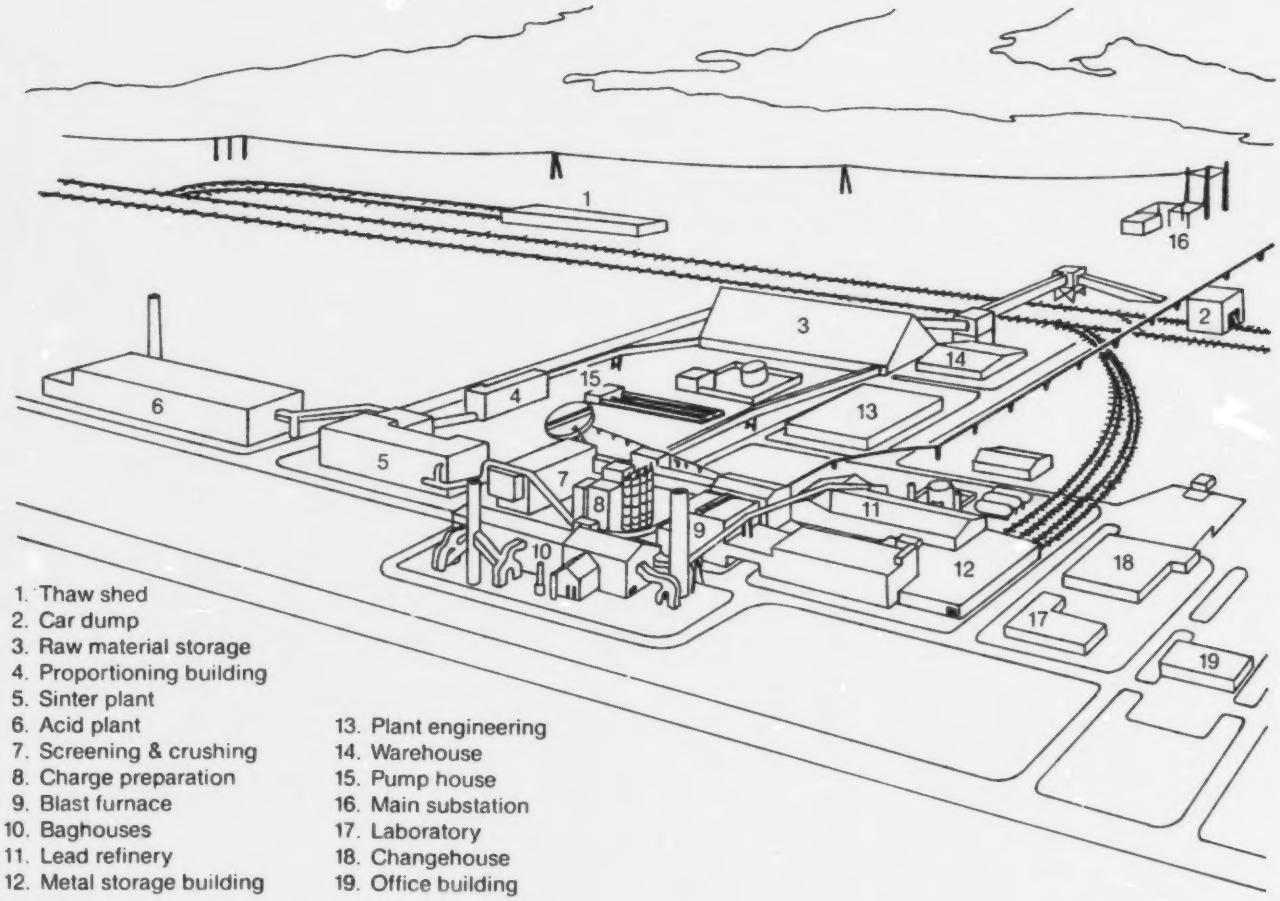
* Given the immobility of lead in sediments, this must have been due to scouring.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1. Thaw shed
2. Car dump
3. Raw material storage
4. Proportioning building
5. Sinter plant
6. Acid plant
7. Screening & crushing
8. Charge preparation
9. Blast furnace
10. Baghouses
11. Lead refinery
12. Metal storage building
13. Plant engineering
14. Warehouse
15. Pump house
16. Main substation
17. Laboratory
18. Changehouse
19. Office building

Figure VIII.2. Belledune smelter complex, NB.

Source: Brunswick Mining & Smelting Corp. Ltd., 1985.

Table VIII.I

Quantities of Contaminants Discharged by the Belledune Smelter Before and After Installation of the New Effluent System

Contaminant	Old system (before Dec., 1980) (untreated and discharged to harbour) (kg/d)	New system (Dec., 1982) (treated and discharged outside harbour) (kg/d)
Copper	1.9	0.5
Lead	23.8	0.72
Zinc	179.0	7.15
Cadmium	70.5	2.12
Arsenic	78.1	17.14
Suspended solids	2440.0	8.80

Source: Hildebrand, 1984; New Brunswick Department of Municipal Affairs and Environment, 1986

These measures were taken in the wake of a significant impact on coastal marine life. Lead, zinc and cadmium levels are raised in various marine biota, as part of a broad contamination pattern within the Baie des Chaleurs, of unspecified origin. Lobster harvesting was restricted near Belledune after the discovery in 1979 that cadmium was being concentrated in a particular organ of lobsters (the tomalley). Lead contamination itself was not a problem. The federal Department of Fisheries and Oceans had maintained a fishing control zone along a 13-km strip of the New Brunswick coast south-east of Belledune Harbour. Lobster harvesting was prohibited in the harbour itself, and special processing of the catch was required in the rest of the zone. Early in 1985, the harbour was included in this zone to permit local harvesting again (Belledune Environmental Monitoring Committee, 1986), the lobster having a marked decrease in cadmium take-up. In 1986 the control zone has been reduced by half, and now includes only a 6 km strip.

This small but significant environmental impact illustrates again a general point about the lead industry: that one cannot mine or process lead without also producing other metallic contaminants, because of the complexity of the ore-bodies. These metals and metalloids, e.g. cadmium, zinc, arsenic and antimony, are often toxic, and in some cases unsaleable. Adequate environmental control demands, therefore, that these by-product metals be disposed of satisfactorily. Cadmium is an especially dangerous element to free into surface waters.

(b) Trail and Kimberley, BC*

As we saw in Section III, the largest primary lead-producing operations in Canada--second in the world--are those conducted by Cominco Ltd. at Kimberley (mining and concentrating) and Trail, BC (smelting, refining and by-products). Both facilities are in deep mountain valleys, with restricted and canalized air flow. Both use much surface water in their operations. As well, both support urban communities potentially exposed to increased concentrations of lead in air, water and soil, unless tight environmental control is maintained.

The operations at Trail have long been in the environmental limelight because of sulphur dioxide emissions. Cross-boundary air flow led to allegations of damage to crops, forests and wildlife in the State of Washington. The International Joint Commission received this case as a binational reference before World War II, and its decision was something of a landmark in environmental regulation. The company's technical response to this decision, and to subsequent regulation by the Province of British Columbia, have created a good deal of awareness in the local community of environmental hazards, and of the need for control measures.

At Kimberley, the Sullivan Mine (see Figure VIII.3) has been in production for over 70 years, and still has 20 years of reserves left (assuming continued production at a rate of about 6500 t/d). It produces lead and zinc concentrates, and smaller quantities of iron (no longer smelted), copper,

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* We are indebted to Cominco Ltd., and especially to Mr. Walter J. Kuit, for help in preparing this section, and to Mr. Kuit and Mr. Graham Kenyon for organizing our comprehensive and open site visit of the Trail facility. We also appreciate the help of the Kimberley Chamber of Commerce, the Mayor of Trail (Mr. Charles Lake) and Locals 9672 and 9705 of the United Steelworkers of America. Cominco's briefs to the Commission were a superb contribution to our work.

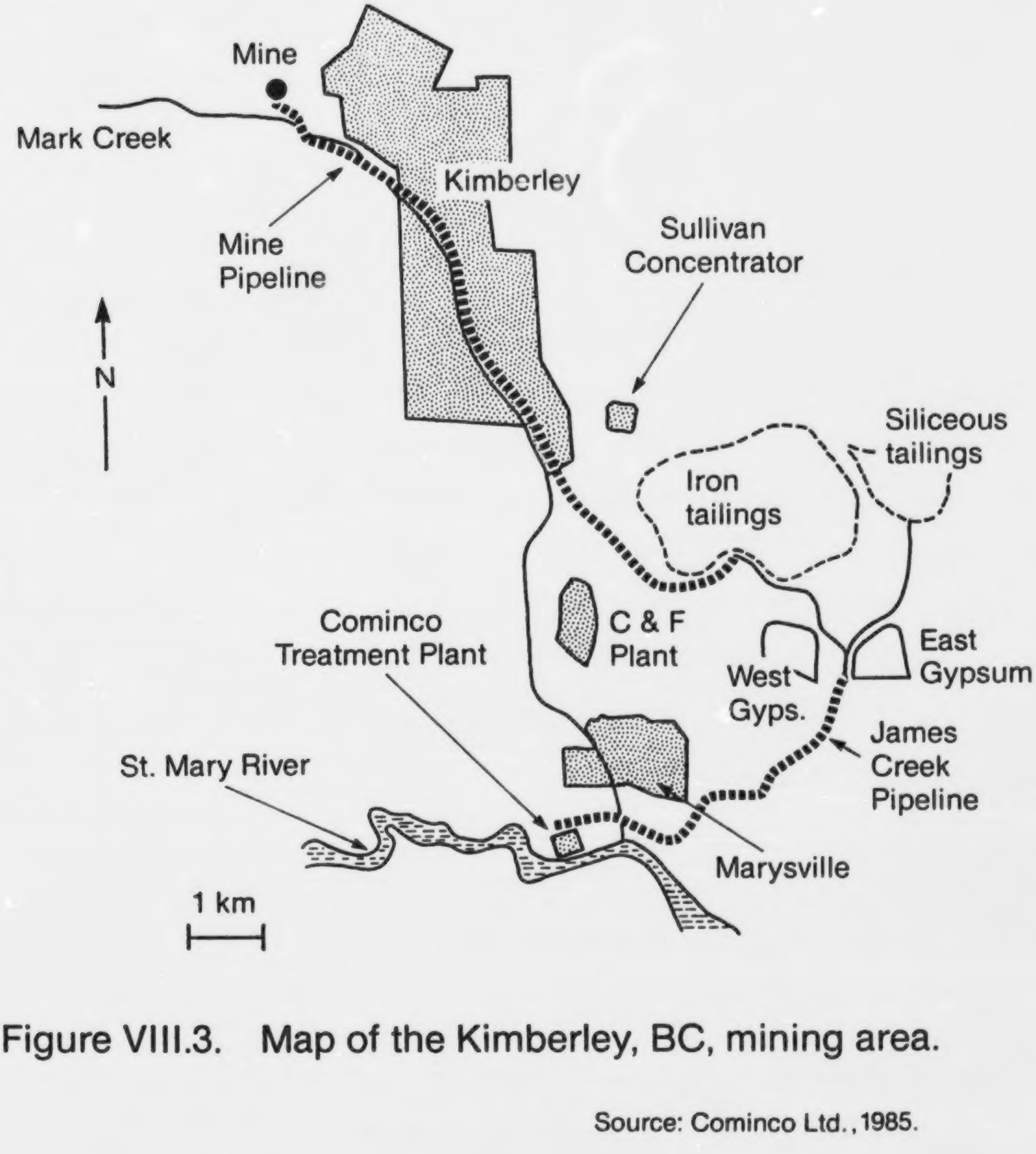
Mine
Mark Creek
Kimberley
Mine Pipeline
Sullivan Concentrator
N
Siliceous tailings
Iron tailings
Cominco Treatment Plant
C & F Plant
East Gypsum
West Gyps.
James Creek Pipeline
St. Mary River
Marysville
1 km

Figure VIII.3. Map of the Kimberley, BC, mining area.

Source: Cominco Ltd., 1985.

silver and tin. The ores contain pyrrhotite, which creates two hazards, both now well contained: acid mine drainage and tailings effluents, and underground fire. Reduction of the effluent problem to provincial standards was achieved by a plant built in 1979. 1984 production is summarized in Table VIII.II. The total recovery of pure lead in this process of concentration was 108 800 t (compared with 84 700 t of zinc). The lead and zinc concentrates are shipped to Trail (200 km west) by rail.

The metallurgical complex at Trail (Figure VIII.4) stands in the dry, deep valley of the Columbia River, which threads a tortuous path southward towards the US border, 10 km distant. The plant and townsite are situated on one of the few areas of flat, well-drained ground available. Stream flow, ground-water movement and air flow are all constricted by the surrounding hills.

The plant accepts concentrates from the Sullivan Mine, and also smelts concentrates and ore from many other Canadian and foreign sources. In its years of operations, the Trail plant has produced (to December, 1984) 9 435 667 t of lead, 9 098 848 t of zinc and smaller quantities of silver and gold. The sulphur dioxide derived from sintering the sulphide ores is sold commercially, and also provides input to a fertilizer operation that has produced 18 448 248 t to the end of 1984. The complex uses coal, oil and natural gas as energy inputs, as well as coke and hydroelectricity. Outputs include a huge volume of slag (largely inert) and quantities of arsenic and antimony (some of the latter being incorporated into lead alloys).

Air emissions and liquid effluents from the complex are governed by permits issued by the British Columbia Ministry of the Environment. The full

Table VIII.II

Production from the Sullivan Mine, Kimberley, BC, 1984

Material	Tonnage	Assays		Distribution	
		% lead	% zinc	% lead	% zinc
Ore processed	2 477 400	5.1	4.0	100.0	100.0
Lead concentrate	176 600	61.6	4.9	86.1	8.6
Zinc concentrate	170 000	4.1	49.8	6.3	85.0
Combined tailings and float rock	2 130 800	0.45	0.30	7.6	6.4

Source: Cominco Ltd., 1985

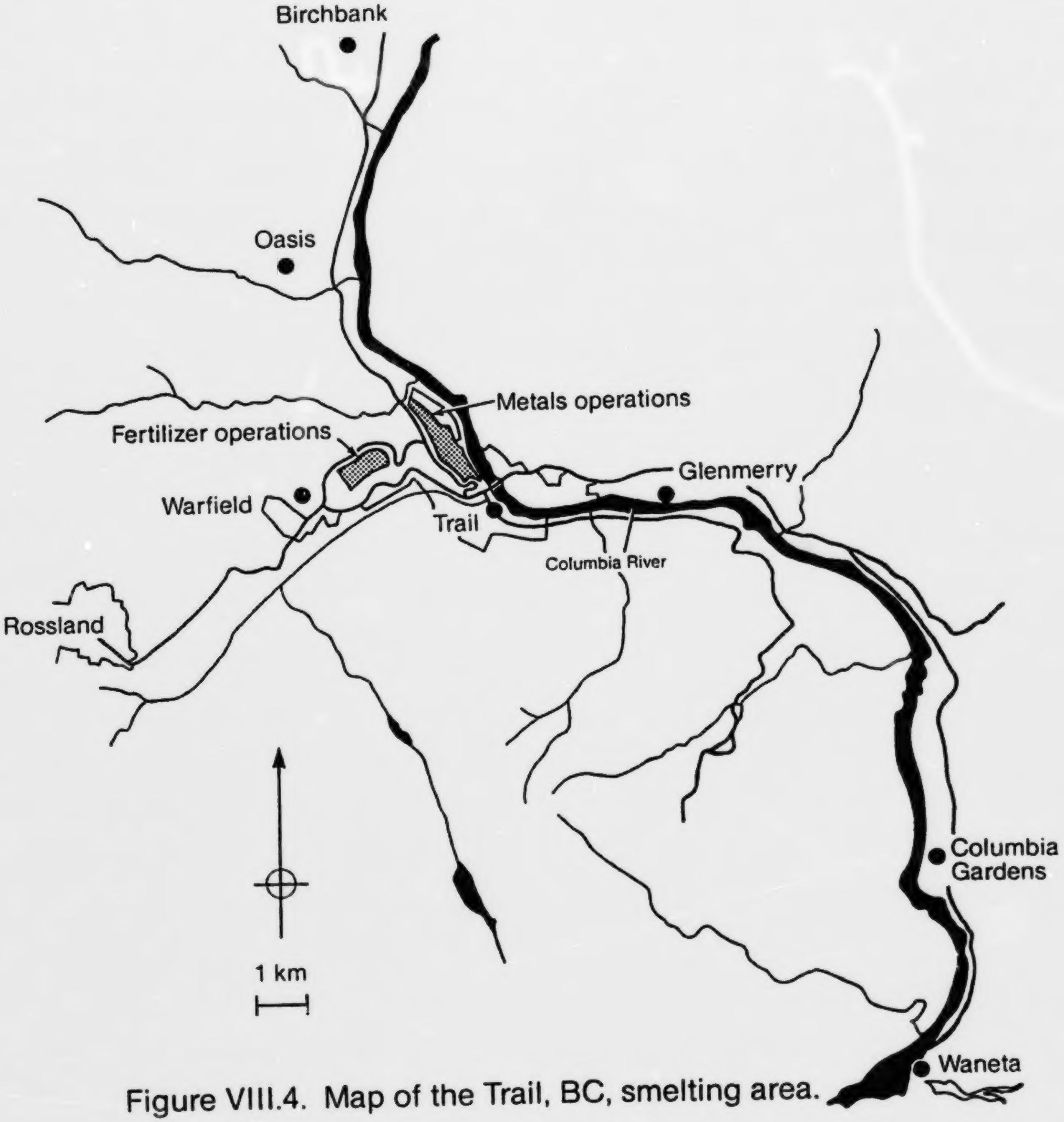
Birchbank
Oasis
Metals operations
Fertilizer operations
Warfield
Glenmerry
Trail
Columbia River
Rossland
Columbia Gardens
Waneta
1 km

Figure VIII.4. Map of the Trail, BC, smelting area.

Source: Cominco Ltd., 1985.

conditions of these permits are not being satisfied, not so much because of failures in the pollution control technology but because the plant is old; the technology is well below the levels of efficiency now metallurgically possible. The items regulated include sulphur oxides, lead, zinc, cadmium, arsenic, fluorides and total particulates among the emissions, and suspended solids, lead, zinc, cadmium, arsenic and mercury among effluents to the Columbia River. Tight monitoring of ambient concentrations and releases is required by the permits.

Lead emissions in 1984 were 140 t, of which over half came from the sintering operation. Liquid effluents are discharged by four sewers to the Columbia River. In 1984, lead effluents totalled 94 t, compared with 628 t of zinc. Table VIII.III shows the measured impact of the plant in 1984, by means of a comparison of parameters measured above and below the sewer discharges. The net effect for dissolved lead is negligible, with a small increase in zinc concentrations. The federal requirement governing total metals (and not merely dissolved) is inapplicable, because Trail was in operation prior to 1977.

Blood lead screening of the local population in 1975 (Neri et al., 1978, 1984) showed that children aged 1-3 and 6 years had average levels of 22 µg/dL, compared with a level of 14 µg/dL in nearby Nelson, a town of comparable size and site, but without metallurgical industry. Older children, less exposed by play habits to dust, differed little in blood lead levels between the two towns. Adult workers in the plant showed blood lead levels in the range 16 µg/dL (office workers) to 41 µg/dL (directly exposed to lead), which is typical of industrial exposures.

Lead deposition onto the soils of this long-established plant has naturally been considerable. Concentrations are highest near the complex, and

Table VIII.III

Columbia River Water Analyses - 1984

(Above (Stoney Creek) and below (Waneta) Trail discharges)

Parameter	Stoney Creek	Waneta
Temperature (°C)	9.4	9.6
pH	7.8	7.9
Conductivity (µmol/cm)	155.0	166.0
Hardness (µg/L)	65.0	70.0
NH_3-N	0.1	0.1
Sulphate (µg/L)	10.6	15.8
Fluoride (µg/L)	0.09	0.16
PO_4 - total (µg/L)	0.01	0.04
Mercury - total (µg/L)	0.00005	0.00005
Iron - dissolved (µg/L)	0.07	0.03
Lead - dissolved (µg/L)	0.02	0.02
Zinc - dissolved (µg/L)	0.02	0.04
Copper - dissolved (µg/L)	0.01	0.01
Arsenic - dissolved (µg/L)	0.005	0.005
Cadmium - dissolved (µg/L)	0.005	0.005

Source: Cominco Ltd., 1985

decrease away from the plant wall. Schmitt et al. (1979) showed that child blood lead levels also decrease with distance from the plant. Table VIII.IV shows the correlation in a series of residential areas progressively closer to the plant. Blood surveys have not been repeated since the mid-1970's, but we received no statements of concern from local sources, including physicians in local practice. Even when we asked them directly, they replied that they had no reason to be concerned about health effects of lead.

The experience in this hot spot in many ways parallels that at Belledune, a smaller and younger installation. Emissions and effluents have been brought under control by large capital expenditures on technical equipment and by disciplined management. Local anxieties have been largely allayed. Blood lead levels among children were high in the mid-1970's, but have since probably fallen (there are few observations). Public fears are now about the viability of the industry. These have been partially answered by the offers of the Governments of Canada and British Columbia to purchase preferred shares from Cominco Ltd. to help modernize the lead smelter, using advanced technology. Stage I of this welcome step is to go forward. The reopening of the Cypress-Anvil complex in the Yukon Territory will feed more lead into a situation of oversupply. Unease thus persists, and has kept environmental concerns in the background.

5. The Problem of Decaying Paint

In striking contrast with US experience, the Commission received no hard evidence that paint was a significant source of lead uptake. Indoor flaking paint has been seen as a main cause of high blood levels in US inner cities (and some isolated rural cases). No evidence of such effects exists for

Table VIII.IV

Lead Concentration in Surface Soil and Children's Blood by Residential Area of Trail, British Columbia

Residential area(s)	Mean soil lead concentration (μg/g) ± standard error (and no. of samples)	Blood lead concentration (μg/dL), mean ± standard error (and no. of children)	
		1- to 3-year-olds	Grade one children
1 and 2	225 ± 39 (26)	17.2 ± 1.1 (27)	18.0 ± 1.9 (18)
5	777 ± 239 (12)	19.7 ± 1.5 (11)	18.7 ± 2.3 (12)
9	570 ± 143 (11)	20.7 ± 1.6 (19)	19.7 ± 1.0 (16)
3, 4, and 8	1674 ± 183 (53)	27.7 ± 1.8 (14)	23.8 ± 1.3 (31)
6 and 7	1800 ± 212 (51)	30.2 ± 3.0 (16)	25.6 ± 1.5 (26)
TOTAL	1320 ± 212 (153)	22.4 ± 1.0 (87)	21.9 ± 0.7 (103)

Source: Schmitt et al., 1979

Canada, though several briefs to the Commission have suggested that an impact must exist, especially in the older cities.

The case of the Weston school yard has been described above. Large flakes of paint were visible in the debris removed from school windows (see Figure VIII.1). Since the removal of this paint it has been shown, however, that the major source of lead in dust in the school yard is gasoline emissions. We have no evidence that past contributions of this paint seriously affected blood lead levels in children.

Ontario blood lead surveys have shown some evidence of peeling paint (not necessarily leaded). In Toronto, in 1984, 9.7% of the subjects' bedrooms had frankly peeling paint, and 14.9% some peeling. In playrooms the corresponding proportions were 3.7 and 19.4% (Ontario Ministry of Health, 1985). On the other hand, only 7% of homes assessed in the city were wooden (compared with 50% in the NHANES II analysis) (Rachlis and Hertzman, 1986); exterior paint has been suggested as a source of intake in some recent US analyses.

The Commission speculated that the towns and cities of the Maritime Provinces, which have more wooden houses, might have experienced this problem. At a meeting of Directors of health services from Nova Scotia and New Brunswick held in Halifax in February, 1986, however, we were told of no case where paint was a significant factor, though some of our respondents had been medically trained in major US cities where lead paint is a problem. Similar opinions were expressed to the Commission Chairman in St. John's, Newfoundland, where no problem had been encountered.

In South Riverdale, Toronto, just over half the children identified in 1983 and 1984 as having blood lead levels above 20 µg/dL lived in houses with peeling paint (Rachlis and Hertzman, 1986). These authors felt that this may be a factor in their elevated blood lead levels, but could offer no further evidence.

The lack of information on paint as a hazard in Canada arises chiefly from the lack of clinical evidence of lead toxicity among children; where no clinical harm is visible, few local health authorities are likely to invade peoples' homes in search of leaded paint that is very likely to have been overpainted, or scraped off and removed.

We found little awareness across the country that paint removal itself may be a hazard. Though the lead content of paint for toys, furniture and interior paints has been regulated under the Hazardous Products Act since 1972 (see Section III), lead oxides (chiefly red lead) continue to be used in paints designed to protect steel structures against corrosion. It is unlikely that this process directly exposes human subjects to significant burdens (subject as always to the possibility of occupational exposure). Old paint, however, is stripped off before the structures are repainted, often with dry abrasives. This creates a cloud of coarse-grained dust, which will be deposited locally.

The medical consultant to the Centre hospitalier de Verdun, Dr. Jean-Guy Bonnier (1986), reported to us that the periodic repainting of the Mercier Bridge across the St. Lawrence River caused just such a problem. This stripping and repainting operation takes place every 10 years, a figure typical of good maintenance procedures for steel structures. In the summer of 1984, a stripping took place. The paint removed contained 15% (150 000 μg/g) lead. Dry sand-blasting was used. Subsequent monitoring showed that:

- the dust deposited on Boulevard LaSalle beneath the bridge (which spans several streets, as well as the St. Lawrence River) was assayed at 16 000 μg/g lead
- air lead concentration in September, 1984, was measured at 1.8 $\mu g/m^3$
- dust removed from inside a nearby house in July, 1984, contained 8000 μg/g of lead

After complaints from nearby residents, work was suspended, and the responsible Minister subsequently ruled that lead paint would not be used in the maintenance of such structures in future. Throughout urban areas, moreover, future paint removal and the cleansing of structures would require the use of wet sand-blasting.

Dr. Bonnier pointed out to us that there are 351 known structures of this sort in Quebec alone, and that a wide variety of owners, operators and public authorities were responsible for their upkeep.

6. Remedial Action and Lead Control for Hot Spots

(a) Decommissioning and Clean-up of Old Sites

The Commission is not expert in the question of decommissioning industrial sites, but strongly supports the moves now under way in both federal and provincial governments to establish guidelines and eventually regulations governing this necessary process. Severe contamination of industrial sites by lead and other heavy metals, as well as organic toxins, has left a legacy that needs careful handling. All operators of such sites should be required to prepare a decommissioning plan in order to avoid a perpetuation of the legacy problem. To do so they require clear guidelines or regulations with which to comply.

The options available, as a plant approaches the end of its life, are defined by Monenco Consultants Ltd. (1985) as mothballing, partial plant shut-down, abandonment and decommissioning. Abandonment, too often the course adopted in the past, is environmentally quite unacceptable, especially where toxic materials are left on the site, or are able to escape from it via emissions or effluents. It must be recalled, however, that much of the lead-using or lead-producing industry in Canada is carried out in urban areas by small companies with limited resources. The present depressed state of the industry makes abandonment and bankruptcy a likely fate for the weaker companies. We are sure that all such sites should be properly decommissioned, even where the operators are unable to finance the work. This implies public financing, perhaps along the lines of the US Superfund (established under the Comprehensive Environmental Response, Compensation and Liability Act, 1980, usually called CERCLA).

The Ontario Government's draft 1984 guidelines for the shut-down of industrial sites are an excellent model of what is needed (Rinne, 1986). Applied at present to oil refineries and storage sites and chemical plants (notably waste sites), they specify much of what is required for the lead industry. They also cover railways and other potentially contaminated sites. As noted earlier, high concentrations of lead may occur on the sites of iron and steel plants, in iron foundries using automotive scrap, and in non-ferrous metal plants, as well as in lead smelters and refineries themselves. Moreover, the primary lead industry may produce significant quantities of other toxic metals and materials (such as acid mine waste and cadmium). Hence, the decommissioning of lead plants requires a look at many contaminants other than lead.

Some of the land on which such plants now stand (e.g. the secondary lead smelters) is potentially valuable for other purposes, and may be a significant asset for the company owning it. If the land is to be used for housing, schools or parkland, it is especially necessary to ensure that lead concentrations on the site be reduced to acceptable levels.

In all instances, the final plan for a decommissioning and/or clean-up will have to be developed for the specific site, bearing in mind its past history; its geochemical and other physical/chemical characteristics; the intended land use; and potential for control of land use in the future. An example of this in a rather general sense is already built into the Ontario Decommissioning Guidelines, where suggested action levels are lower for sandy than for clay or silty soils, and where land intended for residential use is required to meet more rigorous standards for a number of contaminants than is land intended for industrial use.

There is general agreement that 1000 µg/g of lead in soil warrants concern for human health (Missouri, 1986). There is, however, considerable divergence of opinion as to the definition of a removal level. Designations such as trigger levels, action levels and removal levels have been used. Clearly, it would be impracticable and probably unnecessary to remove lead-contaminated soil from a waste site which lay miles away from a human settlement. (This is assuming no leaching, and addresses only the direct route from soil.) Therefore, a combination of soil testing and intelligent consideration of potential for human (especially children's) exposure to the soil should be applied. This is especially relevant for situations that are not strictly decommissioning, e.g. old waste sites, or residences close to major highways or point sources. The proposed limits of 1000 μg/g lead in soil to be used for parkland or industrial or commercial development, and 500 μg/g for residential areas currently in use by the Ontario Ministry of the Environment and several other jurisdictions, seem reasonable. It has to be noted, however, that in certain parts of the world, notably in parts of the United Kingdom, soil levels far exceeding the proposed 1000 μg/g occur quite frequently in residential areas, with no significant effect on the blood lead concentration of children (Southwood, 1983). We have already referred in Section VI to the inconsistencies in data relating children's blood lead to soil lead. The lack of a reliable or consistent relationship has resulted in criticism of the 500 or 1000 μg/g limits as being too low. However, in the absence of any universally acceptable model, it seems prudent to use the most conservative estimates of safety for sites where human contact with soil is anticipated.

(b) Soil Removal and Replacement

Soil removal from contaminated residential areas has already been practised on a limited scale. Examples were described above from Winnipeg and the vicinity of the Toronto lead smelters. We believe that such soil contributes significantly to the uptake of lead by children, and that excessive lead concentrations should be removed. Once a site is identified as requiring such clean-up, the details of physical removal, disposal of contaminated soil and acquisition of replacement soil or fill should be part of the plan.

Because a significant proportion of current lead-fall is still from car exhausts, it would be wise to defer such replacements until after the halving of lead emissions expected in 1987, i.e. until 1988.

The situation described above for South Riverdale, which is apparently the site of a comprehensive current study of soil lead/blood lead relationships, may well provide a test case on which subsequent decisions could be modelled. If further soil removal is carried out in the neighbourhood, there will be a unique opportunity to collect technical, environmental and health data in a comprehensive and valuable way.

(c) Public Education: the Role of Community Groups

We have been constantly reminded that there is little public awareness of lead toxicity in Canada, even in the recognized hot spots. Nevertheless, public interest groups have been active in these areas, and have played a notable part in moving towards removing local hazards. All these groups have emphasized public education as a means of lessening exposures. The Niagara Neighbourhood Association has prepared material (see their submission in Report VI) that indicates the approach used recently in Toronto. Though the Commission is not in a position to evaluate these education drives, the approach seems to be sound. The pamphlet "Get the Lead Out" was developed by the South Riverdale Community Health Centre and the Ontario Ministry of the Environment, and published by the City of Toronto Department of Public Health. It presents its material in simple but non-demeaning style, and gives instructions or suggestions about improving safety; these provide reassurance for the members of the community that they have some control over their own situation. If we cannot provide these communities with better surroundings, then they deserve special consideration in terms of education and counselling, as well as technical help to continue the process of environmental improvement.

7. Regulation of Lead in Sewage Sludge

In Section V, reference was made to the application of sewage sludge as a fertilizer on land, and to the range of lead concentrations measured in this sludge. Metals in sewage sludge and their availability to plants are well-studied topics, and will not be discussed in any more detail here. However, in order to provide a fairly complete consideration of lead contamination of soils, brief reference is made to the current regulations concerning sludge application on agricultural land. Metal concentrations in sludge-amended land are limited, either by controlling the amount of metal added or by limiting the concentration in the land that is receiving sludge. It is noted that there are limits for the soil pH as well. Table VIII.V shows the current regulations for Ontario.

The Commission is satisfied that lead in sewage sludge, if controlled as advocated, will not pose a health problem. However, if sewage sludge is provided to individual home gardens, as was the practice in the 1970's in parts of Ontario at least, then testing of the sludge at source would be necessary to protect the consumer. The practice should, however, be discouraged or, better still, prevented, in any areas where sludges are contaminated with metals.

Table VIII.V

Ontario Regulations for Lead in Sewage Sludge

A. For agricultural land receiving sludge:

Maximum permissible soil lead level:	60 µg/g
Maximum addition of lead:	90 kg/ha

B. For aerobic and air-dried (de-watered) anaerobic sludge:

Maximum allowable lead concentration: 1100 µg/g of solids

(application rate is regulated)

Source: Ontario Ministries of
Agriculture and Food, and
Environment and Health, 1986

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IX What Does the Situation Imply for Canada?

This report has dealt with environmental and health problems created by a metallurgical industry that used to be profitable, and may again be so, but which is currently experiencing difficulties. The fall in world commodity prices has included lead, which now sells so cheaply that Canada's mines, concentrators and smelters are in deep financial trouble. Their employees and the towns in which they live are anxious about their jobs, homes and families. The Commission shares that anxiety.

The industry is suffering, especially at the smelting level, from the out-dated status of some of its heavy equipment. The Commission has been inclined to blame this situation on industry's failure to invest adequately during the high profit years of 1973 to 1979. But the smelter operators pointed out to us that the modern, competitive installations now being planned or started (as at Trail) use technology that was not available until the mid-1980's. In any case, further advances in environmental control and in occupational health depend primarily on the new technology--at a time when profits are low, and investors reluctant to find capital.

Overall, and in the longer term, we have not the least doubt that Canada can afford to produce, sell and consume the lead required by her industry, with what she can sell abroad, and still protect her own citizens against the consequences. We have not tried to test this conclusion by economic analysis, but we have at least analysed the impact of the gasoline phase-down. The modelling exercise by DRI referred to in Section IV is published in Report VI. It asked the question: what would be the effect of the phase-down on gross domestic product? It found that the effect would be negligible. There would be small transfers of investment, income and employment between provinces. Otherwise, the economy could absorb the change with few detectable stresses. We have no doubt that the same is true of the place of lead in the national economy. It will continue to be used for years--and protecting the Canadian public against the environmental overspills will not be an unbearable cost. A hard-pressed smelter operator will not see it this way, because of short-term liabilities. But it is none the less true.

The Commission was struck by the way this issue is fragmented by jurisdictional complexities. Lead is not the only example. Every real environmental problem is similarly divided. Democratic governments have a special genius for putting asunder problems that nature has put together. The use of lead is one example.

Everywhere we went, we heard complaints about this sundering of responsibility. At Belledune for example, the Belledune Environmental Monitoring Committee asked us to help sort out the questions: who monitors what, where, and with what means? Who does what in regulating, advising and negotiating? The Committee is itself a brave effort to create some unity in an otherwise inexcusable complexity. We found similar perplexities everywhere.

Federally, it is clear that the problem of lead involves at least the Ministers of Environment, Transport, Health and Welfare, External Affairs, Energy, Mines and Resources, Labour, Supply and Services, and Consumer and Corporate Affairs. Moreover, it involves their departments. Several of the latter collect data, write guidelines and monitor performance. Within each province there is a similar complexity--and the provinces have the jurisdiction in most aspects of these lead questions.

We were commissioned by the federal Minister of the Environment, and address this report to him. But the necessary action to follow up our recommendations clearly lies as well with several other federal ministers and, at the level of monitoring, regulation and enforcement, particularly with several ministers in each province. Accordingly, we have not directed these recommendations to specific ministerial addresses, but will rely on others to do so.

Two major objectives should guide any attempt to rationalize this complexity:

- There will be a continuing market for lead within Canada for many years, and a continuing world demand that should permit a high future level of exports. To compete in this highly competitive market, the industry will have to modernize its technology. This is true of both primary and secondary smelters, and of most other divisions of the industry. Proper environmental performance should be an integral part of the investment in the facilities required.

- There is a need in government and industry alike to see this industry as a whole, in terms of what public needs it should serve. The problems of the secondary lead smelters will illustrate the point. To their immediate neighbours they may appear as nuisances or even threats to the health of children. Of course they should not be located, as some now are, on the fringes of residential areas. But an efficient recycling mechanism requires that such smelters exist, and that waste batteries and other waste lead not be permitted to accumulate in storage or in municipal dumps.

In the Commission's opinion, now is the time to try to correct the imbalances and imperfections of the entire lead industry, which in many ways has served Canada well--but at the expense of alienating its own neighbours.

Appendix I

Background and Conduct of Inquiry

by Margaret C. Grisdale

1. Establishment of Commission

A lengthy process undertaken by Environment Canada in the early 1980's to examine the impact of lead in gasoline on human health culminated in controversy and conflicting opinion. Pressure to reduce or eliminate lead from gasoline was increased by the action taken by the United States Environmental Protection Agency (EPA) in 1984 to reduce drastically the lead content of gasoline in that country. The Environment Minister concluded that the advice of an independent body was required.

Discussions concerning the establishment of this independent inquiry began in April, 1984, between Environment Canada officials and the Royal Society of Canada (RSC). The Society was accustomed to serving government in this way. Moreover, it was completely independent, and able to draw upon the expertise of Canada's leading scientists and scholars. The role envisaged for the Society in these discussions was similar to that played by the Standing Royal Commission on Environmental Pollution in the United Kingdom, which has conducted its investigations on a scholarly rather than a public inquiry basis, but which has succeeded in achieving a consensual resolution of conflict over environmental issues.

Detailed negotiations continued throughout the summer of 1984. It was eventually agreed that the Society would undertake a study considerably broader than the investigation of the lead-in-gasoline issue originally identified. The Royal Society of Canada received and considered the invitation of the Government of Canada to form an independent commission to conduct an inquiry into all aspects of lead in the Canadian environment, with the exception of lead in the workplace, and, on August 8, 1984, gave its final acceptance. It was further agreed at that time that Dr. F. Kenneth Hare would chair the study, and that Margaret C. Grisdale, his Executive Assistant at Trinity College, would manage it. Other members of the Commission would be appointed by the Royal Society on the recommendation of Dr. Hare.

Though firm agreement between the Society and the Government of Canada was reached on August 8, a delay in the issuance of a contract and the release of funds resulted from the impending election and subsequent change of government in early September. A firm contract, valued at $545,246.00, was awarded to the Royal Society on September 7, 1984, with a final delivery date of September 30, 1987. In addition to the items covered by the contract, Environment Canada agreed to provide office space for the Commission, to print its reports and to cover the salary and benefits of its Chief Scientist under the Executive Interchange Programme. The Chairman and Manager began work on the investigation immediately the contract was signed, working from Trinity College. Suitable premises for the Commission were not found and occupied until November 11, 1984.

The single most important criterion for the selection of Commissioners was expertise. The Chairman and the RSC officers endeavoured to identify the areas in which expertise was required, and then to find qualified individuals for appointment. Eventually appointed were:

- an environmental biologist and heavy metals expert (Dr. Stokes, the Vice-chairman)

- a toxicologist (Dr. Butler)
- a chemist with extensive experience in the mining industry (Dr. Gauvin)
- an engineer with a background in the petroleum industry (Mr. Keough)
- an epidemiologist (Dr. Neri)
- a physician and surgeon (Dr. Robertson)

Three of these six were Fellows of the Royal Society. The absence of a Commissioner with a background in the neuro-developmental field was subsequently felt. Dr. Peter L. Rosenbaum, a pediatric neurologist associated with McMaster University, was identified and appointed as senior medical consultant. He worked very closely with the Commission, particularly the Health Sub-committee, and his knowledge and judgement proved to be invaluable.

In addition to its Commissioners, engaged on a per-diem basis, and its Chairman and Manager, engaged on the basis of 20% of Trinity College salary (payable to the College), two full-time and two part-time staff were employed: Dr. Marcus C.B. Hotz as full-time Chief Scientist officially as of December 15, 1984, Ms. Angelica Devito as full-time Administrative Assistant as of December 6, 1984, and Ms. Kartini Rivers and Ms. Carol Murray, appointed later on a part-time basis to work in the Commission office as word processor operator and research assistant, respectively.

2. Method of Inquiry

The Commission adopted a scholarly approach to the assessment of evidence, but recognized that it must take into account the views of industry and special interest groups as well as the published and unpublished views of research scientists. The budget, in fact, included $50,000.00 to assist health and environmental groups to prepare submissions to the Commission. It further recognized that the evidence gathered must be assessed from the Canadian viewpoint, and that any recommendations it might make would probably have economic and social implications. With these rather formidable requirements before it, the Commission went about its business of gathering and assessing evidence in a variety of ways.

The regular business meetings of the Commission were, of course, essential. The full Commission was assembled at these and, in the early stage of the investigation, devoted considerable time to identifying the task and ways in which it might be executed. It advised the Chairman on the appointment of consultants, planned the programme of the workshops and public meetings and provided direction on the aspects of the issue to be identified. As time went on, specific questions were discussed in detail, and the preparation of the Commission's reports was reviewed. The Commission endeavoured to meet in regions where the lead industry was prominent, or where concern over lead pollution had surfaced. To avoid duplication of travel, business meetings were combined with site visits. Thirteen regular business meetings, one organizational meeting and two drafting sessions were held in all, and these took place in Bathurst (New Brunswick), Edmonton, Montreal, Ottawa, Sarnia, Toronto and Vancouver.

In addition to full business meetings, groups of Commissioners met as required, and individual Commissioners visited and worked in the Commission office. The work of the Health Sub-committee, composed of Dr. H.R. Robertson as Chairman, Dr. G.C. Butler, Dr. L.C. Neri and Dr. P.L. Rosenbaum, is notable in this respect. Assessment of the health effects of low-level lead exposure was probably the most challenging of the Commission's tasks, and in an attempt to grapple with it the Health Sub-committee held numerous meetings in Ottawa and Toronto, often asking outside experts to join them or consulting such experts by conference telephone call. In addition, Dr. Neri used part of his leave from the University of Ottawa to work full-time in the Commission office from April 18 to June 21, 1986, to complete the review of the statistical and health evidence. His services were provided entirely without remuneration, and he claimed only his travel and living expenses.

Among the Commission's first tasks was that of making its existence known. Paid advertisements were placed in the Globe and Mail national edition on January 24, 1985, and in La Presse on January 25, 1985, to announce the establishment of the Commission and its terms of reference and composition, and to invite written submissions. At about the same time, press releases were issued in both official languages making a similar announcement. The releases were sent to all on a list of media contacts that was prepared immediately, and that was maintained and refined throughout the inquiry.

As a further means of contacting interested parties, the Chairman began, in early December, 1984, to write individual letters in which he invited written submissions from federal and provincial government departments and agencies, municipal governments, industries, interest groups, labour organizations and medical associations. Well over 100 such contact letters were sent. The result of these solicitations and other direct contacts made as the inquiry progressed was the 66 submissions listed in Appendix II. The scope and depth of analyses of these submissions varied considerably, but many were well documented, well researched and highly relevant to the Commission's inquiry.

Another initial task, and one that continued throughout the inquiry, was the acquisition of relevant articles, documents and other resource material. With the assistance of Carol Murray, the very large volume of material collected was catalogued and filed. The University of Toronto Institute for Environmental Studies Resource Centre was most helpful in conducting literature searches for the Commission. Government organizations and industry proved to be most generous in providing background information, and very few books and documents actually had to be purchased. A complete bibliography of the literature identified and/or obtained is presented in a separate volume.

The Chairman, Commissioners and Chief Scientist drew upon their extensive network of contacts in the national and international scientific community to promote the work of the Commission and to seek facts. Potential sources of information and interested parties consulted included:

- Sir Richard Southwood, Chairman of the UK Standing Royal Commission on Environmental Pollution, Dr. Donald Chant, President of the Ontario Waste Management Corporation, Dr. Mitchell Perry, a specialist on the lead/blood pressure connection from St. Louis, and Dr. Sergio Facchetti, the Italian scientist who conducted the Turin isotopic lead experiment, all of whom were invited to meet the Commission in Toronto
- Dr. Craig D. Stevenson, of the recently established New Zealand Lead Commission, who spent a day in Toronto with the Chairman and Chief Scientist on July 2, 1985
- the US EPA in New York, Washington and Research Triangle Park with which the Chief Scientist conducted two series of meetings, between February 25 and 28, 1985 and again on May 28, 1985.

- the Commission of the European Community in Brussels visited by the Chief Scientist in May, 1986
- the UK Royal Commission on Environmental Pollution, the UK Ministry of Agriculture, Fisheries and Food and the UK Department of the Environment in London visited by the Manager in January, 1985
- the Philadelphia Health Commission visited by the Manager in May, 1986
- scientists in Italy and other parts of Europe, visited by Drs. Neri and Butler in April, 1985
- members of Imperial College and the UK Ministry of Agriculture, Fisheries and Food in London visited by Dr. Stokes in January, 1986
- the World Health Organization, Geneva and the Centers for Disease Control, Atlanta with which Drs. Neri and Robertson established contact

The Commissioners and staff took advantage of visits to the United States and Europe on other business to seek information on behalf of the Commission. Many of the above contacts were made on such trips.

In his capacity as Chief Scientist, Dr. Hotz represented the Commission on many occasions, including attendance at a hearing of the House of Commons Standing Committee on National Resources and Public Works and two meetings of the City of Toronto Board of Health. He kept in touch with the Justice Department Study on Compliance With Regulations and the Treasury Board Study on Regulatory Reform. He attended numerous workshops and conferences, among them a workshop conducted by the Air Pollution Control Association in Toronto on April 22 and 23, 1985, a seminar sponsored by the Electrochemical Society on battery technology on May 8, 1985, a meeting of the EPA Clean Air Scientific Advisory Committee in Washington from May 13 to 15, 1985, and the Energy, Mines and Resources meeting on alternative fuels in Ottawa on June 10 and 11, 1986. His site visits included the Canadian Oil Company waste lubricating oil re-refinery in Toronto and the Ford Motor Company in Oakville. He also undertook specific responsibility for convening a meeting of Directors of health services of the Maritime Provinces and staff members, in Halifax in February, 1986.

Commissioners and other staff also attended conferences, made site visits and conducted regional meetings. Dr. Neri accompanied the Chief Scientist to the Duke University Conference on Lead and Environmental Health in May, 1985. Dr. Stokes attended the Imperial College Conference on Lead in the Home Environment in London in April, 1986, and presented a paper on soil removal at a conference on trace substances in Columbia, Missouri, in June, 1986. All or part of the Commission visited the following industrial sites:

- the Esso Refinery and Research Centre in Sarnia (January, 1985)
- the Cominco smelter in Trail (October, 1985)
- the Canada Metal Company Ltd. and the Toronto Refiners and Smelters Company Ltd. in Toronto (January, 1986)
- the Brunswick Mining and Smelting Corporation mine near Bathurst, NB and their smelter in Belledune, NB (February, 1986)

In addition, the Chairman and Manager visited Winnipeg in January, 1986, to meet the Deputy Environment Minister, to receive a comprehensive presentation from officials of the Manitoba Environment Department and to tour the Canada Metal Company Ltd. plant and the Weston School area.

The Commission endeavoured to meet those with an interest in the lead issue in the various regions that it visited. Labour and community representatives of the Trail/Kimberley regions of British Columbia and of the Bathurst/Belledune region of New Brunswick were personally invited to meet the Commission, as were senior management officials of Cominco and Brunswick Mining and Smelting. Attendance at meetings other than these was poor. The Commission's experience of public meetings is described in detail below.

The striking aspect of the meetings with labour, community and corporate officials was the remarkable similarity of the views expressed. The unions emphasized that the health and safety of their workers continued to be a prime concern, but indicated that they recognized that further restrictions on the use of lead, or further controls imposed on mining and smelting operations, could cause industry shut-downs and the resulting loss of jobs. They further recognized that alternative employment, where it existed, would provide substantially lower salaries. These views were shared by the community leaders, who recognized the economic vulnerability of one-industry towns. The representative of the Kimberley Chamber of Commerce explained that a tourist industry was being developed as an alternative to mining, but that salaries offered in it were very much lower than those in mining. The Cominco and Brunswick Mining and Smelting officials stressed that the metals market was generally depressed, and that the lead market in particular was in a slump; the companies could not afford to bear any further restrictions on the use of their product, nor the costs of additional environmental controls.

In collecting and assessing information, the Commission did not fail to take account of the resources of Environment Canada, other federal government departments and provincial government departments. The Commission took the view that its arms-length relationship did not preclude availing itself of the information and expertise available within it. Consequently, members of federal government departments were invited to present papers at the Health Effects and Pathways Workshops; observers from Environment Canada, Health and

Welfare Canada and other federal government departments were present at other workshops and public meetings; and the expertise and information available within Environment Canada were consulted.

The Commission distinguished between experts, in government and elsewhere, whom it contacted and consultants, the latter term being applied to those who were paid for their services and advice. A broad range of relationships existed within this category. The close association of Dr. Rosenbaum with the Commission as its chief medical consultant has already been mentioned. Other individuals and organizations, in addition to those who presented papers at workshops, were:

- Prof. David Hewitt who helped assess the epidemiological evidence
- Dr. S.H. Zlotkin who reviewed the biochemical analysis of the report
- Dr. Arthur Chamberlain who, in addition to presenting a paper at the Pathways Workshop, visited Toronto twice to advise the Commission, and to help prepare its final report
- Graduate students of the University of Toronto and of the University of Ottawa who were employed as junior consultants to assist with research and documentation
- ESSA (Environmental and Social Systems Analysts Ltd.), which was employed to enlarge upon its lead model of the city of Toronto in connection with the Pathways section of the report
- DRI (Data Resources of Canada), which was employed to undertake an analysis of several options with respect to the phase-down of the use of lead in gasoline

DRI was selected after a great deal of careful consideration of the type of economic advice required by the Commission: complex advice on economic matters was required, and the huge data base of DRI was drawn upon to provide a comprehensive overview of the economic repercussions of the Commission's recommendations. The fraction of the total budget assigned to consulting fees was by far the largest, because the Chairman and the RSC recognized that it would be impossible to encompass within the Commission the variety of expertise required, and that advice and information would have to be sought elsewhere as required.

3. Workshops

The Commission also recognized soon after the beginning of its investigation that it needed to be educated in specific aspects of the lead issue, and that the way to acquire this education was through a series of workshops rather than commissioning consultants' reports. Six workshops were held: two (Health Effects and Pathways) were organized entirely by the Commission, and the other four were organized entirely or in part by industry or industry representatives. Workshop topics, organizers and dates were:

- The Technical Aspects of Petroleum Refining (organized entirely by PACE, Sarnia, January 17 and 18, 1985)
- Medical and Epidemiological Evidence (studies commissioned by ILZRO, Toronto, February 18, 1985)
- The Tetraethyl Industry (presentations made by DuPont Canada Inc. and Ethyl Canada Inc., Toronto, February 18, 1985)
- Health Effects (the Commission, Ottawa, March 29 and 30, 1985)
- The Metal Industry (presentations by industrial users of lead, co-ordinated by Ms. Darlene Varaleau of The Canada Metal Company Ltd., Toronto, April 26, 1985)

- Pathways (the Commission, Edmonton, October 17 and 18, 1985)

Oral presentations at these workshops were supported by binders of written material, and the exchange among the presenters, Commissioners, staff and invited observers was most valuable. As mentioned elsewhere in this report, the papers and overviews of the Health Effects and Pathways Workshops are presented as separate volumes.

The Commission's only disappointment with respect to the workshops was its failure to conduct, or have conducted on its behalf, a workshop on the automobile engine, focussing on the workings of the internal combustion engine, the promise of the lean-burn engine and the response of automobile designers to existing and impending pollution controls, including the control of lead pollution. Unlike other industries and industry groups approached for information, the Canadian Motor Vehicle Manufacturers' Association (CMVA) was not able to assist. Only after the Chairman wrote directly to the presidents of the five major Canadian automobile manufacturers did the CMVA indicate a willingness to comply with the Commission's request. It then proved to be impossible to agree upon a date and format that would meet the Commission's requirements.

Officials of the Ford Motor Company later proved to be very helpful in obtaining adequate information on automobile engine design to complete the study. The Ford Motor Company of the UK had provided fully illustrated information on the workings of the internal combustion engine to the UK Royal Commission on Environmental Pollution that was generously shared with the Royal Society Commission. In addition, through personal contact, Mr. Keough and Dr. Hotz were able to meet Mr. Ron Bright and colleagues at the Ford Motor plant in Oakville on December 10, 1985, at which time the Commission's many questions with respect to engine design were answered.

4. Public Response to the Work of the Commission

The over 100 contact letters sent by the Chairman, the Commission's advertising in the public press on a national scale, the issuing of two press releases, media coverage resulting from the public meetings and release of the Commission's interim report resulted in surprisingly little response from the general public. Of the 66 formal submissions received, only 6 were from private individuals, one of which was partly financed by the Commission, and only 10 were from special-interest groups, 6 of which were fully or partly funded by the Commission. The majority of submissions were from federal, provincial and municipal government departments and industries.

Brief consideration was given by the Commission to conducting a public symposium on the lead issue, at which representatives of all interested parties would be brought together. The idea was abandoned because of lack of enthusiasm on all sides.

In its place a series of five public meetings was conducted to ensure that all individuals, groups and industries with an interest in the lead question were given an opportunity to make their views known to the Commission. These meetings took place as follows:

- July 25, 1985, in Toronto
- October 4, 1985, in Ottawa
- October 19, 1985, in Edmonton
- December 18, 1985, in Vancouver
- March 25, 1986, in Montreal

The July 25 and October 4 and 19 meetings were advertised in the national edition of The Globe and Mail and in La Presse on June 25, 1985. The December 18 meeting was advertised in the Vancouver Sun and Province, the Victoria Times-Colonist and Le Soleil de Columbie and the Trail Daily Times on November 15, 1985. The March 25 meeting was advertised in the Montreal Gazette and La Presse on February 25, 1986. In addition, the Commission's presence on the east coast and its willingness to meet individuals or groups interested in the lead question in their regions or to pay transportation costs for a meeting in a central location were advertised in the national edition of The Globe and Mail, the Fredericton Gleaner, the St. John's Telegram, the Halifax Chronicle-Herald, the Charlottetown Guardian and Patriot and L'Acadie Nouvelle on January 16, 1986.

Attendance at the meetings and response to the extensive and expensive advertising were disappointing, to say the least. Two interest groups were heard from at the July 25 meetings, both of which had made written submissions to the Commission: a representative of South Riverdale Community Health Centre appeared in person, and the Learning Disabilities Association of Canada (LDAC) asked that a statement be read into the record of the meeting. A representative of the LDAC appeared in person at the October 4 meetings, as did a representative of the Canadian Institute of Child Health. Representatives of only one group attended the October 19 meetings, the Alberta Association for Adults and Children with Learning Disabilities (AAACLD).

The December 18 meeting proved to be the most stimulating and informative: presentations were made by Richard Gallagher on behalf of the British Columbia Medical Association Environmental Health Committee, and by Joe Labuda, P.Eng., who offered a detailed critique of the Commission's interim report, "Lead in Gasoline: A Review of the Canadian Policy Issue". An interesting exchange took place among the Commissioners, invited presenters and observers in attendance from PACE, Cominco and the Vancouver Board of Health and representatives of the various media.

The Montreal meeting was poorly attended by the public and by public interest groups: STOP and the Quebec Association Against Acid Rain indicated a desire to address the Commission, but only a representative of STOP appeared at the meeting. In attendance, however, were representatives of the Canadian Oxygenated Fuels Association, the Montreal Urban Community, le Centre hospitalier de l'Université Laval and le Centre hospitalier de Verdun. At the specific invitation of the Commission, presentations were made on the potential of oxygenated fuels and on the problem resulting from flaking leaded paint on the Mercier Bridge in Montreal. Also present as observers were representatives of the Montreal General Hospital and Environment Canada.

No requests whatever to meet the Commission resulted from the advertising in the Maritime newspapers.

The views urged upon the Commission by the LDAC were that lead was an extremely harmful substance, that it adversely affected the cognitive development of children and that it should be eliminated from gasoline and all other industrial uses at any cost. This view was corroborated by the Canadian Institute of Child Health, which was also of the opinion that children lacked advocates in such issues as the lead controversy. The views of the South Riverdale Community Health Centre, and its particular concern with "hot-spot" problems, are, of course, fully presented in their briefs that are contained in a companion volume to this report. The presentations by Messrs. Gallagher and Labuda at the December meeting, though not fully accepted by the

Commission, were thoroughly researched and well documented, and served to provoke further analysis and discussion by the Commission. The presentation of STOP at the March meeting related only indirectly to lead, and focussed on NO_x in automobile exhaust.

Despite the fact that little new scientific information was received at the public meetings, the meetings served as a useful forum for bringing the various parties to the issue together on an informal basis. The meetings were attended by representatives of PACE (with the exception of the Montreal meeting), Environment Canada, Cominco and others mentioned above with respect to the December and March meetings, and these observers participated in the general discussion that followed the formal presentations. The meetings also served a useful purpose in that they generated media interest in the work of the Commission.

Media representatives were in attendance at all but the October 4 meeting. Most indicated that they had been unaware of the existence of the Commission before the announcements of the public meetings, and that they had not seen either of the Commission's two press releases. Moreover, this lack of awareness existed among representatives of the major media. The release of the Commission's interim report and the Environment Minister's announcement of his strategy to deal with misfuelling also generated media interest in the work of the Commission. The latter aroused the most interest, and the Chief Scientist was called upon to conduct several radio and television interviews as a result. On the whole, media coverage of the work of the Commission was not extensive, and other environmental issues that arose during the enquiry, such as the Great Lakes water quality, received more attention.

The release of the interim report, "Lead in Gasoline: A Review of the Canadian Policy Issue", on October 29, 1985, resulted in some response from the public. An average of 10 telephone calls per day from members of the general public were received until about November 8, 1985. Most of these had to do with misfuelling, suggesting that interest had been sparked more by the Minister's announcement concerning misfuelling immediately before the release of the report than by the report itself.

As mentioned earlier, an amount of $50,000.00 was carried in the budget to assist those without access to other sources of funding to prepare briefs for the Commission and to appear before the Commission. The Commission's willingness to provide financial assistance in suitable cases was clearly indicated when the public meetings were announced. However, there were few claims upon the fund. A brief by Friends of the Earth, a brief by the Niagara Neighbourhood Association and two briefs by the South Riverdale Community Health Centre were financed from it. Also financed from it were the travel expenses of the LDAC representative to attend the October 4 meeting, the travel expenses of the Trail and Kimberley labour and municipal leaders, the costs of having Mr. Labuda's brief typed and photocopied and a small honorarium to STOP for its presentation on March 25. The lack of competing claims for these funds is yet further indication of the lack of public interest in the lead question.

5. Report Preparation and Release

Each Commissioner was assigned responsibility for a specific aspect of the inquiry, and requested to draft the section of the report dealing with that area. The Chairman, Chief Scientist and Manager also assumed specific research and writing responsibilities. The Chairman, with the assistance of the Vice-chairman and Chief Scientist, edited the various individual sections and undertook to prepare a comprehensive, coherent document. This was circulated among the Commissioners, and reviewed and revised at a lengthy drafting session. The revised version was then subjected to Royal Society of Canada peer review before final editing, word processing and French translation. During the drafting stage, an attempt was made to contact those who had made major contributions to the inquiry to ensure the accuracy of the material quoted.

Peer review is standard practice of a Royal Society of Canada inquiry to certify that its reports conform to the original objectives of the inquiry and that scholarly standards were maintained in their preparation. The RSC Fellows assigned originally to review the Commission's reports were Dr. André Barbeau, Dr. Keith Laidler and Dr. Thomas Hutchinson. Sadly, Dr. Barbeau died suddenly on March 9, 1986. He was replaced by Dr. B.R. Belleau. Dr. Derek Bewley was added to the panel in July, 1986, to review the Pathways volume.

In keeping with Royal Society policy to conduct all of its affairs in public, the interim report was released at a press conference in Ottawa on October 29, 1985. The Society and the Commission suffered some embarrassment in that the release of the interim report was delayed for several weeks to accommodate the wishes of the Environment Minister.

6. Liaison with the Royal Society of Canada

Throughout the inquiry, the Commission submitted copies of its regular monthly progress reports to government and to the Society. In addition, the Chairman and Manager met the Honorary Executive Director and the Executive

Secretary on an average of once every 3 months to discuss the work of the Commission.

7. Conclusion

The Commission very quickly discovered the difficulty in avoiding a public hearing type of inquiry in favour of a purely scientific investigation in an issue as politically charged as the lead controversy. It took seriously its verbal mandate to attempt to bring together the various factions and to arrange a consensual plan of action. The degree to which it achieved these objectives is difficult to judge, in that the controversy over leaded gasoline was resolved by the amendment by the federal government of automobile emission control standards that made the use of catalytic converters mandatory and the use of lead as an octane booster impractical. None the less, it achieved a remarkable degree of co-operation from the various participants, and, it is probably fair to say, generated a considerable amount of goodwill.

Its scientific conclusions are stated elsewhere in the report, and it will be for others to judge their soundness. Most serious inquiries face the need to establish boundaries for information gathering, and this was no exception. Given the Commission's resources, in both money and time, it is of the opinion that the investigation was taken as far as was reasonable.

The work of the Commission proceeded without administrative complication, thanks to the excellent co-operation provided by the Administration and Finance Section of the Environmental Protection Service of Environment Canada, and the Contract Officer, Mr. Glenn Allard. Contract payments were made expeditiously, and the officials with whom the Commission worked were unfailingly courteous and willing to provide clarification of the terms and conditions of the contract as required.

The Commission plans to request comments on this final report and on its work in general by November 15, 1986. It will consider these comments, and will circulate them with its own response by December 31, 1986, at which time it will suspend operations 9 months ahead of schedule. Every indication is that the work will be completed within budget.

Appendix II

Written Submissions to the Commission

GOVERNMENT - FEDERAL

Consumer and Corporate Affairs Canada
Energy, Mines and Resources Canada
Environment Canada,
  Environmental Protection Service
Environment Canada,
  Freshwater Institute
Environment Canada,
  National Water Research Institute, Canada Centre for Inland Waters
Environment Canada,
  Transportation Systems Division, Industrial Programs Branch
Health and Welfare Canada

GOVERNMENT - PROVINCIAL

Department of Environment, Newfoundland
Department of Health, Newfoundland
Department of Municipal Affairs and the Environment, New Brunswick
Ministry of Environment, Alberta
Ministry of Environment, and Workplace Safety and Health, Manitoba
Ministry of Environment, Ontario
Ministry of Health, Ontario
Ministry of Environment, Saskatchewan

GOVERNMENT - MUNICIPAL

City of Toronto, Department of Public Health
City of Trail, British Columbia
Communauté Urbaine de Montréal
Regional Municipality of Ottawa-Carleton
Sarnia Lambton Economic Development Commission
Village of Jacquet River

INDIVIDUALS

Clague, Michael
Hubley, Kenneth
Ingraham, T.R.
Labuda, Joe (L.C.E. Company)
Ray, A.K.
Stevens, Harold

INDUSTRIES AND INDUSTRY GROUPS

American Can Canada Inc.
Brunswick Mining and Smelting Corporation
Canada Metal Company Ltd.
Canadian Battery Manufacturers' Association
Canadian Oil Company Ltd.
Canadian Oxygenated Fuels Association
Canadian Paint and Coatings Association
CarterChem Inc.
Cominco Ltd.
Dominion Colour Company Ltd.
Du Pont Canada
Ethyl Canada Inc.
Gulf Canada Products Company
ILZRO (International Lead Zinc Research Organization, Inc.) with individual papers from researchers included in HWR
PACE (Petroleum Association for the Conservation of the Canadian Environment)

INTEREST GROUPS

Alberta Association for Adults and Children with Learning Disabilities
Belledune Environmental Monitoring Committee
Canadian Environmental Law Association
Chaleur Regional Industrial Commission Inc.
Friends of the Earth
Kimberley Bavarian Society and Chamber of Commerce
Learning Disabilities Association of Canada
Niagara Neighbourhood Association
Ontario Association for Children with Learning Disabilities
Society For Environmental Control of Kimberley, British Columbia
South Riverdale Community Health Centre
STOP (Incorporated name; acronym of Society to Overcome Pollution)

MEDICAL ASSOCIATIONS

British Columbia Medical Association
Canadian Medical Association
College of Physicians and Surgeons of Manitoba
Manitoba Medical Association

MUNICIPALITY - HOSPITAL

Centre de Toxicologie du Québec, le Centre hospitalier de l'Université Laval
Département de Santé communautaire, le Centre hospitalier de Verdun

UNIONS

Canadian Labour Congress
United Steelworkers of America, Locals 9672 and 9705
United Steelworkers of America, Local 7085

UNIVERSITIES

Byer, P.H. and R. Serafin, Institute for Environmental Studies, University of Toronto
Nicholls, D. McEwen, Department of Biochemistry, York University,
Wixson, B. G., University of Missouri-Rolla

Appendix III

Octane and its Importance to the Efficiency of the Gasoline Engine

by W.J. Keough

1. The Four-stroke Internal Combustion Engine

During the 20th century, the four-stroke internal combustion engine, fuelled by petroleum gasoline or lighter petroleum products, has become the main form of automotive power for automobiles and light-duty trucks. The engine works on the principle of mixing air and fuel vapour before the mixture is inducted into the engine power cylinder. The homogeneous fuel-air mixture is then compressed, and the compressed gas is ignited by an electrical spark to initiate combustion of the fuel and produce the power stroke of the cycle. Almost all the combustion products are exhausted to the atmosphere. Only small amounts are retained on the surfaces of the engine or in the exhaust system and the lubricating oil.

2. Relationship of Compression Ratio and Engine Efficiency

The first recognized application of the four-stroke cycle engine is generally credited to Otto, who began building engines in the 1880's. His engines exploited the concept of compressing the fuel-air mixture before igniting the mixture. It was readily apparent that the efficiency of his engines could be improved by increasing the compression of the fuel mixture prior to ignition. The success of this type of engine initiated further research into higher compression, and the search for new fuels.

Compression of the fuel-air mixture is expressed as a compression ratio, which is essentially the ratio of the volume of the cylinder at the bottom of the intake stroke to the volume of the cylinder at the top of the compression stroke. The volume of the cylinder includes the volume of the cylindrical portion plus the volume of the head portion, which contains space for the intake and exhaust valves and the electrical spark plug.

In the pursuit of increased engine efficiency, designers at first simply increased the compression ratio. At the same time, petroleum distillate, or gasoline made from the simple distillation of petroleum crude oil, became the preferred fuel for motor vehicle engines.

The combination of higher compression ratio and simple distillate fuel very soon met a technological barrier, a phenomenon known as knocking, a condition that caused a decrease in the efficiency of the engine. If continued, this knocking caused severe mechanical damage. Engine knock could occur with simple petroleum distillates at compression ratios well below 5:1.

3. Fuel Knock Limits Engine Efficiency

Knock is generally attributed to defects in the process of combustion that occur in the cylinder after the spark has ignited the fuel mixture. Under ideal conditions, the combustion initiated by the spark spreads progressively across the combustion chamber until all the fuel is consumed, and a steady, uniform increase in pressure in the combustion chamber and cylinder produces a very efficient power stroke.

When knock occurs, the ideal combustion sequence is interrupted by the premature ignition of end-gas fuel (usually the furthest from the spark plug). The mixture is ignited by a variety of causes, depending upon the condition of the engine, compression ratio, atmospheric conditions, altitude and other factors. Many forms of knock have been identified.

The premature detonation of the fuel can be caused by advancing the timing of the spark, by hot surfaces in the cylinder or by the auto-ignition of fuel at elevated temperature and pressure. The tendency of an engine to knock can be mitigated by using a fuel with a high resistance to detonation.

4. Research Aimed at Solving Knock

Engine designers and petroleum fuel manufacturers began concurrent research to improve the efficiency of engines by changing combustion chamber shape, adding knock inhibitors to fuel and improving fuel molecular antiknock quality.

The earliest and most significant technological development was the discovery by Madgley and Kettering of General Motors Research that organic lead compounds (lead alkyls), when added to petroleum fuel in very small quantities, had the effect of inhibiting end-gas ignition and thus increasing the allowable compression-ratio for a given fuel.

The organic lead compounds not only gave a very significant increase in the antiknock quality of fuel, but did so at a cost that was much lower than any distillation or conversion process developed by the petroleum industry at the time. Lead alkyl antiknock compound became a major technological advance that allowed engine designers to increase engine efficiency dramatically.

5. Octane Quality is Antiknock Quality

As antiknock quality became important in fuel specifications, it was necessary to develop a test to measure this characteristic of the fuel. The test in use today makes use of a single cylinder engine, with variable fuel, load condition and compression ratio, and measures the antiknock quality of a test fuel against a known mixture of iso-octane and normal heptane.

From this test method, the quality of a fuel to resist knocking has been identified as the octane quality. The name derives from the arbitrary assignment of a value of 100 to pure iso-octane fuel.

Two octane measurements are used to characterize motor fuels. Separate test procedures measure the Research Octane Number (RON) (ASTM Research Method D2699) and the Motor Octane Number (MON) (ASTM Motor Method D2700). Each test employs specific conditions of compression ratio, engine load, spark setting and air temperature.

A third octane characterization is calculated from the RON and MON test results. This characterization is the arithmetic average of RON and MON, and is usually referred to as (R + M)/2 octane. It is also occasionally referred to as Road Octane Index (ROI).

6. Engine Design Change to Take Advantage of Higher Octane Fuels

With the availability of lead antiknock compounds in the 1920's, engine designers were freed to begin development of more efficient motor vehicle engines. Concurrent improvements in refining technology also added to the antiknock quality and volume of petroleum fuel available.

Designers immediately increased compression ratios to take advantage of the new antiknock or octane quality. Research into combustion characteristics also led to major changes of the combustion chamber to improve further antiknock performance. One of the major changes was the development of the L-Head engine, which remained popular for over two decades.

In 1925, the typical automobile engine operated at a compression ratio of 4.4:1 on a fuel of 60-70 octane. By the mid-1960's, when efficiencies, compression ratios and raw engine power reached a peak, the compression ratio reached 10:1 and octanes (RON) reached 100.

Production of fuels to meet the demands of the engine designs of the 1960's required petroleum refiners to add new process equipment and in general to use lead alkyl antiknock compounds to the maximum limit of 4 cm^3 per imperial gallon for premium-grade fuels.

Developments in engine compression ratio and fuel efficiency were indeed impressive in the four decades from 1925 to 1965, as shown in Table Ap.I. Horsepower generated for a given size of engine more than tripled.

Table Ap.I

Compression Ratios and Engine Performance

Year	Compression ratio	Horsepower per cu. in. displacement	Engine performance (ton-miles/imp.gal.)
1925	4.4:1	0.234	28.8
1965	10.0:1	0.78	64.2

7. Problems in the 1970's and 1980's

In the 1970's and 1980's, the priorities of engine power and efficiency have been superseded by problems created by the massive emissions of the automobile engine. Initially, concern centred on the hydrocarbon, carbon dioxide and nitrogen oxide emissions from engine exhaust, and their effect on atmospheric pollution and smog formation. Subsequent concern developed on the emission of lead from the exhaust and the increase in lead in the ambient environment.

As we have seen, the use of lead alkyls in motor fuel has been reduced to varying degrees in most countries during the last decade. Motor fuel octanes and compression ratios have declined with decreased use of lead antiknock compounds. Engine designers have been challenged to develop improvements in combustion chamber design and fuel injection to maintain engine efficiency.

Coincident with the reduction in lead and octane, the world also experienced an energy supply crisis that further emphasized the need for engine efficiency, compression ratio and octane. Artificially high prices for oil prompted a retrenchment in fuel use. Thus, research is continuing by both the engine manufacturer and the petroleum refiner to produce engines with improved combustion characteristics and fuels that possess high octane quality without the use of lead antiknock compounds. In many countries, gasoline consumption has actually declined.

Appendix IV

Forecasts of Lead Emissions from Vehicles

We reproduce in this appendix two submissions that predict future vehicular lead emissions in Canada, on the basis of the pending 1987 regulations:

- material from the Inventory Management Division of the Program Management Branch, Environmental Protection Service, Environment Canada, submitted by Frank Vena, June, 1985 (see Tables Ap.II and Ap. III)
- material from PACE, June, 1985 (see Table Ap. IV)

We are grateful for this help. The data have been used to prepare Figure IV.1.

If the Government of Canada proceeds to reduce lead in gasoline to 0.026 g/L (or to eliminate it altogether) in 1992, the emissions will, of course, then drop far below those shown in this appendix.

Environment Canada Environnement Canada
Environmental Protection Protection de l'environnement

Inventory Management Div.
Program Management Branch

Ottawa, Ontario
K1A 1C8

Your file Votre référence
Our file Notre référence

June 3, 1985

Mr. W.J. Keough
c/o Commission on Lead
241 Jarvis Street
Toronto, Ontario
M5B 2C3

Dear Mr. Keough:

In response to your request for information regarding the assumptions and methods used in estimating lead emissions from gasoline powered vehicles, I am providing the following description which should help to answer your questions.

For the years 1933 through 1983, emissions have been estimated on consumption of lead in gasoline. These numbers were provided by Ethyl Canada for the years 1933-1978 and by the refineries themselves after 1978. Further, it was assumed that the lead added to gasoline in each year of interest was also consumed in that year and that during combustion 75 percent of the lead in the gasoline was emitted.

After 1984 emissions are based on projected demand of leaded gasoline and corresponding use. The projected demand figures are derived from an Environment Canada Report "Control Options for Lead Phase-Down in Motor Gasoline" which in turn came from a combination of National Energy Board projections of energy use and Dupont's Escon model predictions of gasoline demand which take into account vehicle type, average number of kilometres driven, fuel efficiency and market demand.

The Dupont model gives a total gasoline demand, i.e. leaded and unleaded as well as a vehicle breakdown for leaded demand (cars, light duty trucks and heavy duty trucks).

* To these projections some modifications have been made in order to include recent regulations governing motor vehicles and gasoline. These are the implementation in January 1987 of a regulation that permits a level of lead in gasoline no greater than .29 g/l and a regulation which stipulates that all new cars sold after September 1987 be equipped with catalytic converters.

In order to conserve energy and resources, this paper contains post-consumer fibre.

À des fins de conservation de l'énergie et des ressources ce papier contient des fibres recyclées.

** The effect of these two regulations on emissions can be seen in Table 1. Here emissions have been calculated based on leaded gasoline demand projections. These have been modified for the years subsequent to 1988 based on a reduction of 10 percent of leaded gasoline burning LDV's each year derived from an estimated 10 year lifespan for the average car and light-duty truck. What this does is remove 10 percent of the leaded gasoline demanded by these vehicles projected to be on the road each year after 1988 so that in 1998 lead emissions are attributed solely to heavy duty trucks. These emission estimates do not include misfuelling.

I will be pleased to address any further inquiries you may have.

Yours sincerely,

Frank Vena

F. Vena
Chief

Attch.

c.c.: Mr. V. Shantora

* Ap.III in this appendix
** Ap.II in this appendix

Table Ap.II

Environment Canada's Analysis and Prediction of Leaded Gasoline Emissions

Year	Lead added (consumed) ($x\ 10^6$ lb)	Calculation	Emissions (t)
1933	(Ethyl) 2.3	(2.3×10^6 lb) (0.454 kg/lb) 0.75	790
1938	3.73	(3.73×10^6 lb) (0.454 kg/lb) 0.75	1 270
1943	5.32	(5.32×10^6 lb) (0.454 kg/lb) 0.75	1 810
1948	7.63	(7.63×10^6 lb) (0.454 kg/lb) 0.75	2 600
1953	11.71	(11.71×10^6 lb) (0.454 kg/lb) 0.75	3 990
1958	15.86	(15.86×10^6 lb) (0.454 kg/lb) 0.75	5 400
1963	21.21	(21.21×10^6 lb) (0.454 kg/lb) 0.75	7 220
1968	32.68	(32.68×10^6 lb) (0.454 kg/lb) 0.75	11 130
1973	43.16	(43.16×10^6 lb) (0.454 kg/lb) 0.75	14 360
1978	26.8	(26.8×10^6 lb) (0.454 kg/lb) 0.75	9 130
1983	(Refineries) 8675.46 t	8675.46 x 0.75	6 507
1988	Leaded gasoline demand 12.53×10^9 L	$12.53 \times 10^9 \times 0.75 \times 0.29$ g/L	2 725
1993	Leaded gasoline demand 7.24×10^9 L	$7.24 \times 10^9 \times 0.75 \times 0.29$ g/L	1 570
1998	Leaded gasoline demand 3.41×10^9 L	$3.41 \times 10^9 \times 0.75 \times 0.29$ g/L	740
2003	Leaded gasoline demand 3.36×10^9 L	$3.36 \times 10^9 \times 0.75 \times 0.29$ g/L	731

Source: Inventory Management Division,
Program Management Branch,
Environmental Protection Service,
Environment Canada, 1985

Table Ap.III

Unleaded and Leaded Gasoline Forecast, 1987-2003

	Base case		New vehicle regulations	
Year	Unleaded ($\times 10^9$ L)	Leaded ($\times 10^9$ L)	Unleaded ($\times 10^9$ L)	Leaded ($\times 10^9$ L)
1980	14.99	23.35	-	-
1981	14.85	22.79	-	-
1987*	23.15	12.94	-	-
1988*	23.15	12.53	23.15	12.53
1993*	22.88	10.94	26.58	7.24
1998*	22.02	10.53	29.15	3.41
2003*	21.75	10.40	28.74	3.36

* All projections of future gasoline demand are based on National Energy Board forecasts made in 1980.

Source: Inventory Management Division,
Program Management Branch,
Environmental Protection Service,
Environment Canada, 1985

Automotive Lead Emissions

Submission by PACE

June, 1985

1. Tonnes of lead emitted (1970-1984) were obtained from Ethyl Corporation Ltd. and correlated with Environment Canada data.

2. Tonnes of lead emitted (1985-1986) were estimated by Ethyl Corporation Ltd.

3. Tonnes of lead emitted were calculated from the formula: (fuel consumption) x (grade split forecast) x (0.29 g/L lead in leaded gasoline) x (75% of lead used is emitted) x (units factor)

    - fuel consumption (billions of litres or 10^9 L) was obtained from Dupont Canada Ltd.

    - grade split forecast prepared from PACE data of May, 1985

    - a constant amount of lead at the January 1, 1987, regulated level was used (0.29 g/L lead in leaded gasoline)

    - lead used is only partially emitted and the 75% factor was used in the Control Option Report and by US EPA

Table Ap.IV

PACE's Estimates of Automotive Lead Emissions

(Fuel Consumption and Grade Split)

Year	Emissions (t) of lead	Gasoline consumption (x 10^9 L)	Leaded gasoline (as fraction of total)
1970	12 523	25.0	
1971	11 774	26.8	
1972	12 414	29.5	
1973	13 938	32.3	
1974	13 332	32.3	
1975	11 954	34.5	
1976	10 883	35.0	
1977	9 668	35.5	
1978	9 136	36.4	
1979	8 958	38.6	
1980	8 228	38.6	
1981	8 110	37.3	
1982	6 806	34.5	
1983	6 492	33.2	
1984	6 461	31.8	0.547 (actual)
1985	6 000	30.9	0.504 (actual)
1986	5 000	30.5	0.46*
1987	2 768	29.6	0.43
1988	2 532	29.1	0.40
1989	2 302	28.6	0.37
1990	2 115	28.6	0.34
1991	1 928	28.6	0.31
1992	1 680	28.6	0.27
1993	1 493	28.6	0.24
1994	1 306	28.6	0.21
1995	1 120	28.6	0.18
1996	1 013	29.1	0.16
1997	901	29.6	0.14
1998	848	30.0	0.13
1999	791	30.3	0.12
2000	663	30.5	0.10

* The actual grade split for the period January to June, 1986 averaged 0.446 (Refined Petroleum Publication Booklet 45-004. Statistics Canada, 1986). The year-end average is now projected to the about 0.435, implying that the change to unleaded gasoline is proceeding much more rapidly than originally forecast.

Appendix V

Experiments to Estimate the Contribution of Gasoline Lead to Blood Lead: Isotopic Lead Experiment - Turin, Italy

An experiment using gasoline lead additive with an isotopic composition sufficiently different from that of background lead was conceived by Facchetti and Garibaldi in 1973, under European Community auspices, as a method of tracing the effects of motor gasoline lead on human blood lead and the environment. The objectives of the project were:

- to determine the contribution of gasoline lead to:
  - ° airborne lead
  - ° blood lead level in various population groups
- to trace the pathways of gasoline lead through the environment

Lead from the Broken Hill mine in Australia, with a $^{206}Pb/^{207}Pb$ isotopic ratio of 1.04, was sufficiently different from that of the Turin area to allow Broken Hill lead to be used in the manufacture of lead antiknock compounds for the area, and to allow tracing of this lead in the environment. The main part of the experiment was conducted from May, 1977, to December, 1979. During this period the lead concentration in local motor gasoline was 0.63 g/L.

Dr. Sergio Facchetti met members of the Royal Society of Canada Commission on Lead in the Environment on June 4, 1985, and presented the latest data from the Isotopic Lead Experiment.

Facchetti noted that new experimental data not yet published tend to confirm the earlier estimates of blood lead contributed by lead in gasoline.

In Turin the contribution is calculated to be 23% in adults, whereas in the countryside within a 25-km radius of Turin it is 11%. He also cautioned that these data apply only to the Turin area population, where adult blood lead concentration in the city is in the order of 23 µg/dL. In areas where population average blood lead is different from that of Turin, the contribution of lead from gasoline will be different. The following boundary conditions have been found to apply:

- conditions that contribute to lower values
    - only 90% of Australian lead was used as an additive in petrols
    - because of the remodelling of bones, the isotopic ratios of blood lead and of the accessible bone lead were likely to converge continuously in an asymptotic manner (the equilibrium had not yet been reached when Australian lead ceased to be used)
    - the first blood sampling of some follow-up subjects was performed shortly after a partial use of the Australian lead in gasoline
- conditions that contribute to higher values
    - the lead concentration in gasoline was about 0.63 µg/L
    - the test area is characterized by very light winds, associated with strong temperature inversions and multiple air stratification, all of which impede vertical mixing
    - the region is a highly industrialized area, with heavy traffic protected on three sides by the mountainous Alpine arc

- the city of Turin has the highest car/inhabitant ratio (0.4 in 1977) of all cities in Europe, and its traffic corresponds to 5.6% of that in all Italy

Table Ap.V compares contributions of gasoline lead to blood lead as originally calculated, and as now revised. Figure Ap.1 shows time series of the main parameters from 1974 to 1984.

Table Ap.V

Comparison of Old and New Measurements on Follow-up Subjects, Isotopic Lead Experiment

Place	Measurements	Number of subjects	Contribution of petrol lead to adult blood lead Mean ± SD	Median
Turin	New	15	0.196 ± 0.131	0.231
	Old	12	0.237 ± 0.054	0.234
	All	27	0.214 ± 0.104	0.231
Countryside < 25 km	New	7	0.084 ± 0.076	0.097
	Old	19	0.125 ± 0.071	0.118
	All	26	0.114 ± 0.073	0.114
Countryside > 25 km	New	6	0.095 ± 0.115	0.092
	Old	4	0.110 ± 0.058	0.087
	All	10	0.101 ± 0.093	0.090

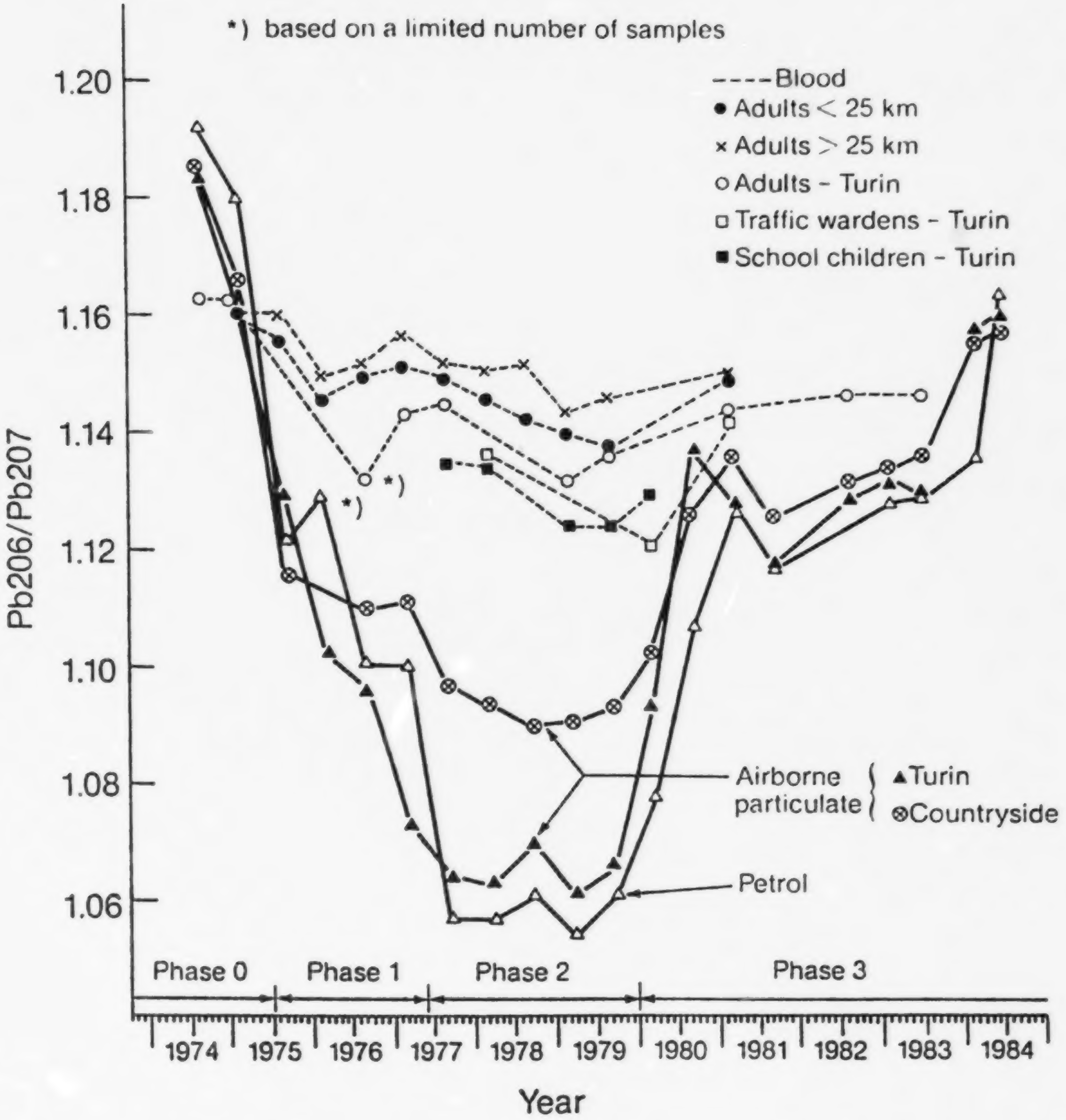

Figure AP.1. Change in Pb-206/Pb-207 ratios in petrol, airborne particulate and blood from 1974 to 1984, Turin area.

Appendix VI

Number of Cases and Deaths due to Selected Poisonings

(As reported to the Product-related Accident Section, Health and Welfare Canada, according to the service required)

(see Table Ap.VI)

Source: Health and Welfare Canada, 1986

Table Ap. VI

Number of Cases and Deaths due to Selected Poisoning Reported to the Product-related Accident Section (LCDC, HWC) According to the Service Required, 1980-1984

Age group 0-4

Attributed cause	1980 (434)*			1981 (396)			1982 (425)			1983 (405)			1984 (405)		
	Phone calls only	Treat. or hosp.	Deaths	Phone calls only	Treat. or hosp.	Deaths	Phone calls only	Treat. or hosp.	Deaths	Phone calls only	Treat. or hosp.	Deaths	Phone calls only	Treat. or hosp.	Deaths
All non-drugs	12 872	10 843	50	19 568	6 470	44	24 348	6 912	51	23 616	7 691	51	22 058	8 017	57
Any drugs**	10 376	8 159	178	12 006	7 147	252	14 273	8 586	203	15 093	8 862	183	14 951	7 941	215
Aspirin	1 383	1 478	5	1 504	2 205	8	2 205	2 118	4	2 846	1 837	0	2 876	1 508	5
Household products	2 944	2 544	6	3 157	1 593	2	4 301	1 671	6	n/a***	n/a	n/a	4 573	1 961	4
Paint chips	34	5	0	54	1	0	58	6	0	53	1	0	64	5	0
Lead	2	2	0	4	0	0	4	2	0	14	2	0	14	5	0

* Number of hospitals reporting.
** Not including aspirin.
*** n/a = not applicable.

Table Ap.VI (Cont'd)

Age group 5-14

Attributed cause	1980 (434)*			1981 (396)			1982 (425)			1983 (405)			1984 (405)		
	Phone calls only	Treat. or hosp.	Deaths	Phone calls only	Treat. or hosp.	Deaths	Phone calls only	Treat. or hosp.	Deaths	Phone calls only	Treat. or hosp.	Deaths	Phone calls only	Treat. or hosp.	Deaths
All non-drugs	1215	1593	0	1959	1052	0	2792	1128	0	2900	1071	0	2070	1500	0
Any drugs**	1137	1173	0	1654	1057	0	1631	1204	0	2822	1311	0	1679	1379	0
Aspirin	180	263	0	232	253	0	236	313	0	208	312	0	209	308	0
Household products	317	189	0	312	146	0	329	169	0	n/a	n/a	n/a	292	216	0
Paint chips	0	0	0	3	2	0	2	1	0	1	0	0	4	1	0
Lead	1	1	0	1	0	0	0	0	0	11	4	0	9	4	0

* Number of hospitals reporting.

** Not including aspirin.

Table Ap.VI (Cont'd)

Age group 15+

Attributed cause	1980 (434)*			1981 (396)			1982 (425)			1983 (405)			1984 (405)		
	Phone calls only	Treat. or hosp.	Deaths	Phone calls only	Treat. or hosp.	Deaths	Phone calls only	Treat. or hosp.	Deaths	Phone calls only	Treat. or hosp.	Deaths	Phone calls only	Treat. or hosp.	Deaths
All non-drugs	1327	860	0	5067	2907	0	5446	3572	0	5562	3877	0	3716	3707	0
Any drugs**	1731	6873	0	5868	6841	0	3015	7477	0	2936	7508	0	2510	7363	0
Aspirin	256	1250	0	465	1067	0	357	1116	0	333	1102	0	302	1201	0
Household products	291	404	0	846	432	0	912	483	0	n/a	n/a	n/a	665	623	0
Paint chips	1	0	0	0	0	0	1	0	0	1	0	0	2	1	0
Lead	2	2	0	2	4	0	4	4	0	6	2	0	3	3	0

* Number of hospitals reporting.
** Not including aspirin,

Appendix VII

Chemistry of Lead

Lead (chemical symbol Pb) is a heavy metal with chemical properties similar to those of tin. It exists in the form of a number of isotopes, which are lead atoms of different atomic mass. The ratios of these isotopes in naturally occurring lead deposits vary according to their history, because three of these isotopes are the final, non-radioactive products of different natural radioactive decay series: lead-206 (^{206}Pb) is derived from the decay of uranium, ^{207}Pb from actinium and ^{208}Pb from the thorium series. The fourth isotope, ^{204}Pb, is of unknown origin. The difference in isotopic ratios of lead mined in different areas was used in the Isotopic Lead Experiment (see Appendix V) to try to identify the proportion of lead absorbed from gasoline, as distinct from other sources, by inhabitants of the Turin area of Italy. Lead behaves as a typical metal, reacting with a variety of acids to form two series of ionic salts, called plumbic and plumbous, according to its oxidation state. The metal is particularly resistant to sulphuric acid.

Lead is easily extracted from its ores. It melts at the relatively low temperature of 327°C, and boils at 1755°C. Like all pure metals, it is soft, but its much harder alloys with tin and copper (the brasses and bronzes) have long been used industrially, architecturally and artistically. Many lead compounds have also proved to be useful industrially. These include a number of oxides, which are commonly used as feedstocks for the manufacture of other compounds.

The inorganic chemistry of lead is straightforward. Important compounds include several oxides:

- PbO	lead monoxide (litharge), yellow or red, often simply called lead oxide
- Pb_3O_4	triplumbic tetroxide (red lead)
- Pb_2O_3	lead sesquioxide
- PbO_2	lead dioxidc, a powerful oxidizing agent, used with metallic lead in the lead-acid battery

Important naturally occurring salts are:

- PbS	lead sulphide (galena), the primary form of lead in ores
- $PbSO_4$	lead sulphate (anglesite), one of the few sulphates that are insoluble in water
- $PbCr_2O_4$	lead chromate (crocoisite), used as the pigment chrome yellow, usually with litharge

Pure water will attack lead, forming the hydroxide $Pb(OH)_2$, which may induce lead poisoning; however, the sulphates and carbonates, often present in natural waters, will form a basic carbonate coating on the lead surface, preventing further attack. Before this coating has formed, however, any lead carbonate produced will dissolve, with consequent risk of toxicity. For many years, carbonates and sulphates were added to municipal water supplies distributed through lead piping in areas with naturally slightly acidic soft water (Parkes and Mellor, 1939).

Lead acetate, $(CH_3COO)_2Pb$, called sugar of lead because of its sweet taste, is easily absorbed in diet, and is extensively used in animal research on lead toxicity. It can easily be made by the action of acetic acid (present in wines, vinegar, and several fruit juices) on lead oxide. It is also an intermediate in the so-called Dutch process used to make the basic carbonate white lead. This compound was the most commonly used white pigment for over

2000 years. Lead arsenate, $PbAsO_4$, was used for many years as an agricultural and horticultural pesticide, until its quite recent replacement by organic toxins. Lead is also found in complex salts of other metals, e.g. as the chloroplumbate, where it occurs in the negative anion of the salt, rather than in the positive cation, e.g. example $Na^+_2[PbCl_6]^{2-}$.

Its atomic structure also allows it to form covalently bonded organic molecules, and its ability to react with organic compounds involved in the biochemical life processes of animals is one of the major reasons for lead's toxicity. Both inorganic and organic lead are toxic; the effects of lead on key metabolic processes are described in Section VII.

Probably the most important industrial organolead compounds are the tetraalkyl lead antiknock compounds used to control the combustion of gasoline vapour in automotive engines. The manufacture and use of these compounds are described in Section IV.

References

Parkes, G.D. and Mellor, J.W., 1939. Mellor's Modern Inorganic Chemistry. Longmans Green, London. p. 703.

Appendix VIII

Authors and Titles of Commission Papers

1. Health Effects of Lead

Lead and Health--An Editorial Overview
Marcus C.B. Hotz

Measurement of Lead
Paul B. Hammond

Methodological Aspects of Graphite Furnace Atomic Absorption Spectrophotometry in Blood Lead Analysis
John D. Hamilton and Jean-Pierre Farant

Lead Contamination of the Canadian Environment
Jerome O. Nriagu

Levels of Lead Exposure in Canadian Populations
Albert Nantel

Blood Lead Levels in Canada: A Comparison with Some Other Countries
Luciano C. Neri with Jocelyne Tessier

The Importance of Various Sources of Lead
Peter C. Elwood

Human Exposure to Lead from Gasoline
Joel Schwartz

Dietary Lead Intakes
Robert W. Dabeka

Childhood Lead Poisoning in the U.S.--An Historical Perspective, and the Current Concerns for the Metabolic Effects of "Low Level" Lead Exposure.
Edward B. McCabe

Metabolic and Cellular Effects of Lead: A Guide to "Low Level" Lead Toxicity in Children
John F. Rosen

The Health Effects of Low Level Exposure to Lead
Herbert L. Needleman

Implications of Current Laboratory Findings for Assessing the Human Hazards of Environmental Lead
Bernard Weiss

Behavioural Effects of Developmental Lead Exposure in Monkeys
Deborah C. Rice and Diane C. Kirkpatrick

Neurobehavioural Effects of Lead
A Summary Review of Cross-Sectional and Longitudinal Studies
Robert L. Bornschein

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Transport of Lead from the Environment to Man
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Appendix IX

Definitions

1. Chemical Terms

Cracking:	chemical processes that break down large petroleum hydrocarbon molecules into smaller ones containing shorter chains of carbon atoms
Reforming:	chemically changing the cracked products into compounds that have the properties needed for fuel or other industrial processes
Isomers:	two or more chemical compounds whose molecules contain the same numbers of identical atoms held together by the same kinds of bonds, but which have different properties due to different arrangement of the atoms
Isomerization:	the chemical processes that cause one isomer to change its molecular structure to that of another, e.g. turning straight chains of carbon atoms into branched-chain isomers having higher octane values
Polymer:	a chemical compound whose molecule is made up by bonding together a large number of identical smaller molecules called monomers (plastics are typical polymers)

2. Epidemiological Usage

Ecological studies provide averaged disease or exposure information at community rather than individual levels. The data are aggregated within geographically delineated areas.

Cross-sectional studies focus on a single measurement of health or exposure within a short time period (more or less contemporaneously). Such studies provide information on both the prevalence of disease and the prevalence of exposure, but give no information about evolution over time.

Cross-sectional studies repeated at intervals are termed **longitudinal**. They measure the incidence of disease, i.e. the occurrence of new cases in the same population.

Retrospective studies classify individuals on the basis of the occurrence or appearance of disease. Case and control groups are usually compared with respect to exposure.

Prospective studies follow selected, currently exposed and non-exposed populations to compare the development of disease over time in both instances. Such studies relate the incidence of disease to historical exposure.

Appendix X

Units

t	(metric) tonne
kt	kilotonne (10^3 tonne)
µg	microgram (10^{-6} gram)
mg	milligram (10^{-3} gram)
g	gram
kg	kilogram (10^3 gram)
µm	micrometre; micron (10^{-6} metre)
m	metre
km	kilometre (10^3 metre)
h	hour
d	day
a	year
mL	millilitre (10^{-3} litre)
dL	decilitre (100 mL; 10^{-1} L)
L	litre (10^{-3} m^3)

Appendix XI

Dissenting Opinion by US Researchers

After an exchange of correspondence, the Commission agreed to publish the following rebuttal by four US biomedical researchers who had made presentations at its Health Workshop in Ottawa in March 1985. This appendix is a rebuttal to sections of the interim and final reports that the Commission made available to them.

The Commission received this rebuttal on September 25, 1986, ten days after the final report was received in the office of the Minister for the Environment. Certain points raised by the four researchers had already been changed in later drafts or were dealt with in parts of the report which the Commission had not sent them. These include:

- the Commission's agreement that there are unmistakable biochemical impacts on the hematopoietic system down to very low blood levels. Indeed, it says so in both reports. The Piomelli et al. (1982) study is cited in the final report.
- the Commission's endorsement of the Government of Canada's decision to virtually eliminate lead from gasoline by 1992. This was not included in the section of the report provided to the authors of this text. This decision was announced by the Minister for the Environment on March 25, 1986.

The Commission will reply to these and other criticisms more fully in its final summary of the discussion in December 1986.

Meanwhile, the Commission rejects any assertion that the evidence was assessed selectively or in an archaic manner, and that its recommendations constitute a disservice to the people of Canada.

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In association with
Albert Einstein College of Medicine

September 24, 1986

Dr. F. Kenneth Hare
Chairman,
The Royal Society of Canada
Commission on Lead in the Environment
241 Jarvis Street
Toronto, Ontario
Canada M5B 2C3

Re: Interim and Final Report (pp. 1-14, 177-234, XXXVii-Xliii): Lead in Gasoline, issued by the Commission on Lead in the Environment.

Dear Dr. Hare:

Four American scientists (Drs. Needleman, Rosen, Schwartz and Weiss) were invited by the Society and Commission to present information in our fields of expertise at the Health Effects Workshop on Lead in Humans on March 29 and 30, 1985 in Ottawa. We were also requested to submit papers to the Commission for inclusion in its final report to the Minister. Recently, each of us received and reviewed the Commission's Interim and Final Report cited above. This joint letter from the four of us offers you and the Commission our collective evaluation of the report.

We find that the report ignores many important scientific findings, fails to recognize the significance of the accumulated toxicological data, and adopts a policy of placing the burden of proof of adverse effects on Canadian citizens themselves. It shunts aside the most compelling priority, prevention. Instead of seeking to protect public health by recommending adequate safety margins, the report indicates that lead toxicity is not a significant problem until clinically overt disease becomes evident. To those of us who are familiar with the signs, symptoms and clinical course of lead toxicity in both children and adults, the Commission's archaic position yields a policy that irreversible consequences are the basis upon which exposure standards are erected. The Commission, however, should be guided by the knowledge that, once children develop overt symptoms, irreversible damage to the central nervous system is probably already present.

Statements in the document, that adverse health effects of lead have not yet been established at blood lead values between 30-50 ug/dl and below 30 ug/dl, are applied to areas where substantial evidence of adverse health effects have been demonstrated in humans. This thesis, developed by the Commission, fails to reflect the current judgement of the scientific community; and, even where appropriate, the Commission suggests that evidence of a clearly recognized health hazard to children and the fetus should not be translated into policy, if the evidence is not beyond any "shadow of a doubt."

The Commission's report seems to us a disservice to Canadian citizens and a retreat from informed regulation. Those of us who participated in the review process perceive insufficient evidence that our efforts were weighed seriously, thoughtfully, and impartially. It appears that we were used to provide a similacrum of objectivity.

A collaborative rebuttal to the text of the Commission's interim and final report, as provided to us, is attached to this letter. We have already agreed in writing (Dr. Hare's letter of September 10, 1986), that this letter and attached rebuttal will be included (without changes) in the Commission's final report to the Minister.

Thank you for your assistance in these matters.

Sincerely,

John F. Rosen, M.D.
Professor of Pediatrics
Head, Division of Pediatric Metabolism
Albert Einstein College of Medicine
(With Drs. Needleman, Schwartz and Weiss).

JFR:mh
cc: The Hon. Thomas Mcmillan, P.C.,M.P.
Dr. Alexander G. McKay
Ms. Barbara McElgunn

Dissenting Opinion by US Researchers

Four US Researchers--J.F. Rosen, H.L. Needleman, B. Weiss and J. Schwartz objected to the treatment given to their presentations of data in the Commission's Report. After an exchange of correspondence, the Commission agreed to publish the following rebuttal by these biomedical researchers.

This appendix is a rebuttal to sections of the interim and final reports that the Commission made available to them. Though the Commission may have come very slightly closer to the consensus that has developed in the public health and regulatory communities in the US, substantial differences persist.

1. Highly Significant Neuropsychological Effects of Lead are Present at "Low Levels"

As a scholarly rather than a policy document, the report requires substantial revisions. For example, the report quotes sentences from the review by Yule and Rutter (1985) out of context. The theme of this review is clearly summarized below: "The ... conclusion is probable but not certain--namely that this lead effect of some 2 to 5 points represents a true causal influence of lead on children's neuropsychological functioning ... The main grounds for suggesting that there is likely to be a true causal effect are four-fold: (i) that the effect is biologically plausible; (ii) that, within studies, a dose-response relationship has usually been found--even after controlling for confounding variables; (iii) that the most rigorous control for confounding variables has failed to remove the effect; and (iv) that there is such good agreement in findings between the large epidemiological studies in Britain, the United States and Germany." (pp. 252)

Furthermore, a recent draft of the US EPA's Air Lead Criteria Document states: "Other evidence confirms that neural dysfunctions exist in apparently asymptomatic children at similar or even lower levels of blood lead ... The lowest blood lead levels clearly associated with other types of altered behavioural performance, both in apparently asymptomatic children and in developing rats and monkeys, generally appear to be in the range of 30-50 μg/dL. However, certain behavioural (e.g. reaction time and reaction behaviour deficits) and electrophysiological (altered EEG patterns, evoked potentials and peripheral nerve conduction velocities) effects indicative of CNS and peripheral nerve functional perturbations have been reported for children at lower blood levels, and are suggestive of a continuous dose-response relationship between lead and neurotoxicity down to exposure levels as low as 25-30 μg/dL or, perhaps, somewhat lower."

Official EPA policy is represented by the following statement for EPA's "Cost and Benefits of Reducing Lead in Gasoline: Final Regulatory Impact Analysis" (EPA 230-05-85-006): "These facts (review of the children's health studies), together with the very small p-value calculated in the joint test, suggest that the combined evidence of cognitive effects at moderate levels of lead exposure should be treated as statistically significant. We conclude that the combined results of available studies of cognitive effect at moderate levels should be taken as evidence of cognitive decrements due to lead." (See pp. IV-41.)

This conclusion by EPA was based upon the determination of a joint p value calculated from the results of 6 studies; the calculated p value was <.005 for lead effects on neurobehavioural parameters. It is clear, therefore, that the preponderance of the data have indicated that higher lead groups evidence lower cognitive ability.

These EPA data, which yielded a combined p value of $<.005$ from the result of six studies, have been further extended by evaluating the combined results of 14 studies. The most recent reports of the Edinburgh study (Fulton et al.), the Athens study (Hatzakis et al.) and the Denmark study (Hansen et al.) were included in this analysis. In addition to the latter studies, the following reports were also included in this analysis: Ernhart et al., 1974; Needleman et al., 1979; Yule et al., 1981; Winneke et al., 1982; Smith et al., 1983; Winneke et al., 1983; Harvey et al., 1984; Shapiro, 1984; Lansdown, 1986; Hansen, 1985; Hawk and Schroeder, 1985; and Schroeder et al., 1985. These combined data sets included extensive neurobehavioural assessments, with control for covariates, in a total of 2234 children.

Power analysis for "small" effects, effect sizes and joint probability were also calculated. The last statistic, the probability of the studies yielding the observed p values by chance, under the null hypothesis was calculated using the Fisher Aggregation technique. One-tailed p values were calculated by taking -2LOGeP of the probability; these were then summed. The sum has a chi square distribution with two N degrees of freedom. The chi square was 110 for 26 df; and the joint probability yielded a p value of 3×10^{-12} or 3 in one trillion. We next calculated the number of unpublished studies with theoretical p values of 0.50 (lead effects on neurobehavioural functioning), which would be theoretically required to raise the p value to $\geqslant .05$. The number was 76. We are very doubtful that there are 76 unknown no-effect studies in the files of investigators worldwide.

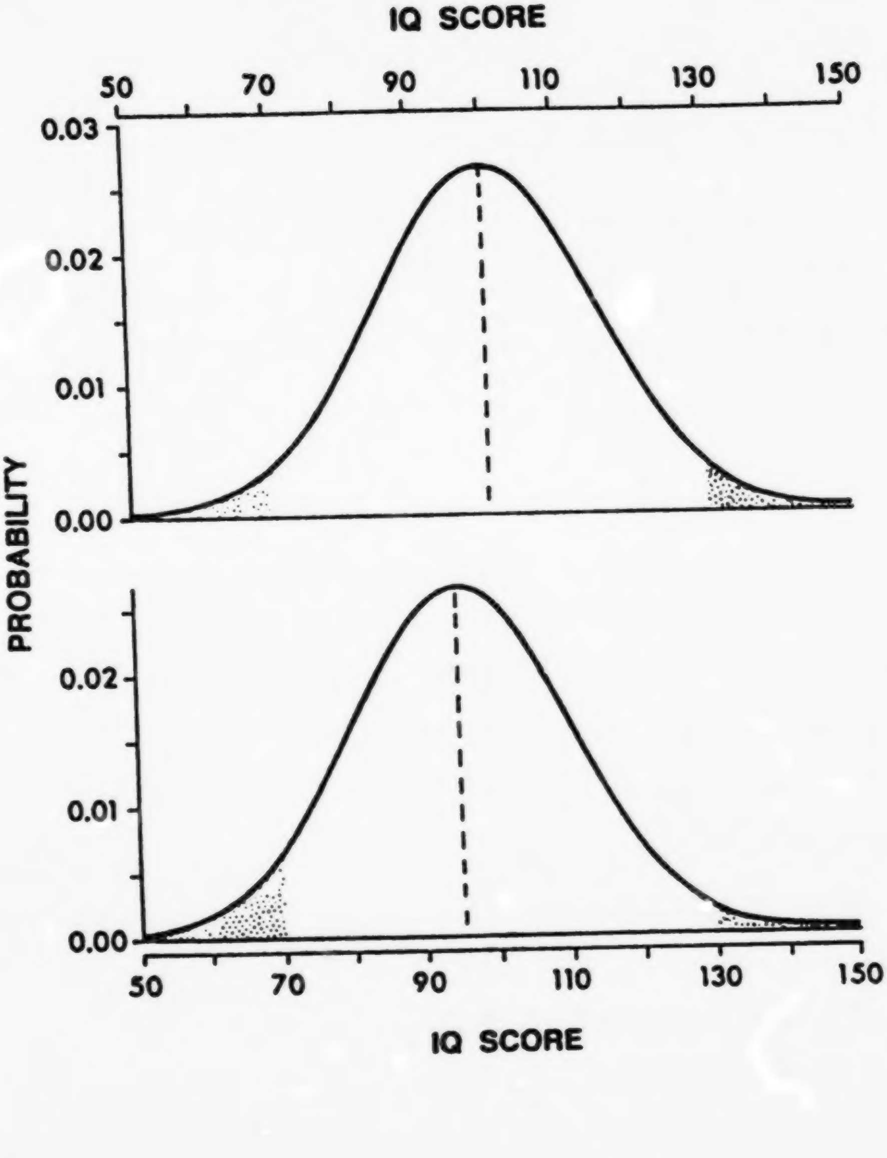
The report claims that definitive answers await the results of prospective studies currently in progress. Yet, the Commission failed to observe that two prospective studies were presented to the Commission in Ottawa. The Boston study, based on a middle class sample, showed that umbilical cord blood lead levels, in a range considered normal, were related to developmental outcome at 6 and 12 months of age. The Cincinnati study showed the same association in its sample of white offspring. It is also puzzling that the Commission's report does not cite the Boston studies, which have been reported (Bellinger et al., 1984, 1986). Since the results of these studies do not accord with its views, some critics might view their omissions as an example of the Commission's willingness to ignore data in conflict with its conclusions.

Several times the Commission's report claims that no conclusive evidence has yet been found for cause-effect relationships between neurophysiological deficits and lead at low levels. At the Ottawa meeting, we discussed at length the limitations of proof in any epidemiological study and, in that framework, scientific evidence was presented indicating that far lower amounts of lead are neurotoxic. As noted above, there is agreement between the Boston and Cincinnati results. Neurobehavioural and electroencephalographic abnormalities, at blood lead levels considerably below 30-50 µg/dL have been noted in many studies, and were also described to the Commission. The Commission owes the Canadian public an explanation for excluding these important results from its final report.

It is perplexing, indeed, to find that the Commission perceives the effects of lead on the central nervous system of children to be "small" (pp. xli). Suppose an investigator asked, that thalidomide, instead of inducing structural anomalies, such as undeveloped limbs, had reduced IQ by 10 percent. Would we have suspected it to be a hazardous agent? The answer to that question is disquieting enough. But consider the data indicating that rather moderate lead exposure reduces performance on intelligence and other psychological tests by perhaps 5 percent. Is it worth being concerned about? A superficial assessment, based on the size of the variation from one test administration to another, which is greater than 5 percent, might argue that it is too small a value to be meaningful. But the implications for society dissolve any complacency such an argument might induce. The figure overleaf compares two IQ distributions. The upper one is drawn with a mean score of 100, the score designed to represent the mean on standard tests such as the Stanford-Binet. The lower is drawn with a mean of 95. The tails of the distribution convey the message. Lowering the mean by a "trivial" 5 percent in a population of 100 million reduces the number of individuals with IQs above 130 from 2.3 million to 990 thousand! At a time when the economic robustness and security of a nation depend, in part, upon its technological resources, that trivial reduction emerges as a societal disaster!

2. Neurobehavioural Studies in Animals: Consistency Between Experimental and Human Findings

One of the more dismaying features of the report is the absence of references to the experimental literature, particularly the more recent research. It is especially dismaying because one of the most significant sources of the newer data is the Health Protection Branch of Health and Welfare Canada. There, studies conducted on monkeys by Dr. Deborah Rice and her colleagues have confirmed and extended the conclusions reached by Needleman and many others from the human data. Her findings demonstrate adverse lead effects at blood levels of 20-25 μg/dL, a range that many


IQ SCORE
50
70
90
110
130
150
0.03
0.02
0.01
0.00
PROBABILITY
0.02
0.01
0.00
50
70
90
110
130
150
IQ SCORE

Canadian children exceed. The epidemiological problems sometimes cited in human studies surely play no role in such results. Moreover, the monkey data parallel data obtained in rats with much the same kind of advanced behavioural techniques and that were described at the Ottawa meeting. The consistency of the human and animal findings surely deserved comment.

3. Toxic Biochemical Effects of Lead are Adverse Health Effects in Children

In view of sections 1 and 2 in this rebuttal, it is rather amazing that the Commission claims that it cannot "establish a point at which lead can be said to harm an individual at low levels of exposure" (pp. xl). In addition to the clinical and experimental neurobehavioural studies, the Commission heard from several laboratories the results of studies indicating that adverse biochemical effects of lead have been demonstrated in children at blood lead concentrations below 25 µg/dL. Moreover, evidence from 4 Centers (Albert Einstein, Hopkins, Columbia and Harvard) demonstrated that a significant percentage of children show positive $CaNa_2EDTA$ provocative test results at blood lead values between 25 to 55 µg/dL. Such data indicate clearly that the blood lead concentration tends to underestimate the body burden and exposure history of lead in American children. They also show that the magnitude of lead excretion in children with blood lead values in the range noted above is similar to that of children with higher blood lead values and who qualify medically for immediate chelation treatment.

It is disappointing that the Commission acknowledged some of the toxic biochemical effects of lead in a rather tentative manner. It ascribed its position to the lack of obvious clinical signs in children showing such biochemical evidence of adverse effects. These Commission views are not supported by the published literature and oppose the judgements, based upon detailed evaluations of the data of regulatory and health agencies in the United States.

The data of Piomelli and co-workers (Proc. Natl. Acad. Sci., 1982) were based upon an analysis of blood lead vs. erythrocyte protoporphyrin in 2004 children, hardly a limited data set. The observed threshold for lead effects (14-17 μg/dL) on erythrocyte protoporphyrin calculated by recognized statistical techniques, has not been challenged. The results of $CaNa_2EDTA$ testing was substantiated independently at four different Centers; and the combined data set in 210 children is by far the largest ever published (Piomelli, Rosen et al., 1984). Lead's capacity to impair the biosynthesis of the vitamin D hormone was revealed in a dose-response relationship encompassing a range of blood lead values between 12 to 120 μg/dL (Rosen et al., 1980; Mahaffey, Rosen et al., 1982). These findings have undergone intense scrutiny in the United States and remain unchallenged by published data. Moreover, such effects of lead on vitamin D metabolism in children have been confirmed directly by experimental studies *in vivo* and *in vitro*, as pointed out at the Workshop (see Smith et al., 1981; Edelstein et al., 1984).

The statement in the Commission's report that it is "premature to conclude that lead specifically suppresses the vitamin D hormone system," attributed to DeLuca (1985), is unfounded. A dose-response depression in circulating levels of the vitamin D hormone in children has been published in the peer-reviewed literature and was presented at the Workshop and was published (Mahaffey, Rosen et al., 1982); and data have been reported indicating that vitamin D hormone levels return to normal after chelation therapy for lead toxicity in children (Rosen et al., 1980). Furthermore, both in vivo and in vitro dose-response inhibition of the 1-hydroxylase enzyme system by lead has been demonstrated and reported (Smith et al., 1981; Edelstein et al., 1984). In the brief statement by DeLuca (1985), it was proposed that a phantom toxic agent other than lead may be responsible for these data in children, in experimental animals and in the test tube. What other toxic agent might this be and why was this reference (DeLuca, 1985) included in the Commission's report? Was the Commission attempting to dismiss the importance of these findings in children? In this regard, it is perplexing why the Commission neglected to include the published results of the analysis by Schwartz et al. (1986) concerning lead effects on the growth of children. For each 10 µg/dL increase in blood lead from 5 to 35 µg/dL, children evidenced a 1.2 cm decrease in height (Schwartz et al., 1986--analysis of NHANES data of height vs. blood lead in children). Presumably, according to the Commission, toxic biochemical effects of lead and lead effects on the stature of children are irrelevant to the growth potential of Canadian children.

Several criteria are readily available to define "adverse health effects" and are well accepted in the United States (see chapter 13 of EPA's Criteria Document). These include: (i) perturbed function of a specific tissue or organ system; (ii) diminished reserve capacity of that tissue or organ system to sustain additional results; (iii) the prevalence of a given effect in a vulnerable group of individuals (such as children and pregnant women): and (iv) the net impact of various perturbations that converge on a single organ or cellular system to impair normal functioning, i.e. diffuse abnormalities in heme or calcium metabolism. The toxic biochemical effects presented to the Commission, which are produced by lead at relatively low concentrations, clearly qualify. These include lead's impairment of heme synthesis, the heme pathway, the cytochrome system, cellular energetics, pyrimidine metabolism, vitamin D metabolism, cellular calcium homeostasis, perturbations in circulating calcium demonstrated in children (see Sorrell et al., 1977; Rosen et al., 1980), calcium dependent cellular processes (including calmodulin activated enzyme systems), the hydroxylation of cortisol in children by hepatic microsomal enzymes and so forth.

This extensive but abbreviated list, and the interweaving convergence of such lead effects on diverse cell types of different tissues are recognized as adverse by the US EPA, the US Public Health Service, and informed pediatricians. This long list also includes lead's impairment of oxygen transport, detoxification of xenobiotic compounds, neurotransmitter functions and depletion of total body heme. The collective impact of these adverse health effects can be avoided by prescribing a generous margin of safety.

It would appear to be reasonable to safe-guard the growth potential of Canadian children!

4. The Relationship Between Lead and Hypertension

The Commission's discussion of the studies relating blood lead to blood pressure suffers from several shortcomings. First, the experimental data is of critical importance, because experimental studies control for all of the host of other factors that are more difficult to manage in a human study. Several different species of animals, exposed to low levels of lead, have all suffered increased blood pressure in multiple experiments by many different laboratories using very strictly controlled protocols. These results remove the question of whether there is causality behind the correlation. Clearly, low level lead exposure causes increases in blood pressure in rats and pigeons.

Given these data, why would one believe that the same would not hold true for humans? Obviously, the only answer is that humans may react differently to lead because they are different; and the blood pressure response may be species specific, rather than related to any general mechanism. The fact that the blood pressure elevation is seen in several species of animals, however, makes that conclusion questionable even in the absence of human epidemiological data. Moreover, since the results of experiments also indicate that the mechanism of lead's effect is in the vascular smooth muscle cells and that lead leads to increased intracellular calcium in the cells of those arteries, the mechanism is identified as general and not species specific. Lead has been shown to cause intracellular calcium accumulation in many tissue types in many species, including arteries, hepatocytes, bone cells and brain tissues; this effect is not one that humans can escape; and change in intracellular calcium homeostasis is the cause of muscle contraction in smooth muscle tissue in general, not just in rats. In particular, it is also true in humans that the higher the intracellular calcium concentration in the smooth muscle cell, the greater is its contraction; and the greater its contraction, the greater is the resistance of arteries to blood flow, and therefore the higher the blood pressure.

In fact, the fastest growing drug for the treatment of hypertension is calcium channel blockers, because they reduce the leakage of calcium into the smooth muscle cells of the vascular periphery, and therefore lower contraction of the cell and constriction of the artery. Since lead does the opposite of the medication used to lower blood pressure in humans, it can hardly be surprising that lead raises blood pressure.

However, these animal studies, which are ignored in the report, are also buttressed by the epidemiological results. These include the papers of Pirkle et al. and Harlan et al., which exhaustively analyzed the NHANES II data and other recent general population epidemiology studies, such as Moreau et al., Kromhout et al., and Pocock et al., whose most recent analysis shows p-values for lead of 0.001 in regression analysis!

Even more disturbing than the omission of these results, which all tend to give a consistent picture, is the Commission's assertion that the results are still not established. The Commission should know, as was eloquently stated by Sir Karl Popper in "Conjecture and Refutation", that no hypothesis can ever be proved true in science; they can only be proven false. When sufficient attempts to prove one false have failed, we accept it until shown otherwise. This acceptance is a gradual and continuous process; and we do not seek to argue over how widely the lead-blood pressure hypothesis deserves to be accepted today. Rather, the question is one of what level of acceptance is necessary, before it is reasonable to take prudent steps to protect public health. This requires a balancing of the costs of being right and the costs of being wrong, as well as the surety of being right. We belive that the abundant animal and epidemiological evidence, demonstrating that lead causes blood pressure increases in animals, and that lead is significantly associated with higher blood pressure in humans, shows sufficient likelihood of an effect. Given the serious consequences of higher blood pressure, it is manifestly imprudent to allow lead to remain in gasoline while there is a good chance that eliminating it will reduce the number of people with hypertension by over 10%.

As noted in EPA's "Costs and Benefits of Reducing Lead in Gasoline" (EPA230-05-85-006, on pp. V-26): "the other low exposure studies, the animal data and the robustness of these results [Lead on Blood Pressure] suggest that the relationship is causal. Moreover, specific analyses to determine whether there is a lower threshold below which lead has no effect on blood pressure showed that the data were fit best with a threshold of zero." Once again, the Commission has failed to protect the health of Canadian citizens.

5. The Relationship Between Gasoline Lead and Blood Lead: Is it the Commission's and Minister's Responsibility to Enhance Public Health at no Cost to Society?

An astonishing omission from the Commission's report is the lack of any substantive discussion of the relationship between gasoline lead and blood lead. This relationship is so strong that it is difficult to reconcile with the conclusion that no further restriction of gasoline lead is necessary. Equally bizarre is the lack of any discussion of the cost savings that will occur if lead levels are reduced, due to the reduction of the corrosive effects of lead on cars, although various costs of removing lead from gasoline are discussed in the Commission's Preamble. These two omissions seriously imbalance the report. Equally incredible in a document specifically directed to gasoline lead is the omission of any reference to or discussion of EPA's analysis of the impact of its gasoline lead regulation, "The Costs and Benefits of Reducing Lead in Gasoline". EPA's Criteria Document, which provides the background for regulation of stationary sources of lead, is referred to, but a 500 page analysis of the exact issue that the Commission is addressing is completely ignored! The Commission was supplied with several copies of this document by the EPA. It is not common practice in science to ignore papers, analyses and data that point to conclusions that one wishes to avoid. The two omissions mentioned above are discussed at length in the document that the Commission refuses to recognize.

The relationship between lead in gasoline and lead in people is well established by both human epidemiology and experimental isotope studies. The NHANES II data showed a strong relationship between blood lead and gasoline lead ($p < 0.000001$), after controlling for age, race, sex, income, family size, degree of urbanization, residence inside center city, region of the country, occupational exposure, smoking, alcohol consumption, dietary food intake, lead used in canned foods, and so forth. The same relationship (also with a t-statistic of 11) was found after analyzing the data from the Chicago blood lead screening program for children, after controlling for race and age. New York's and Louisville's screening programs showed the same relationship; and the CDC data for the national screening program showed a correlation coefficient of 0.80 between the percent of children with high blood lead levels and gasoline lead! Isotope studies by Manton and Terra in the United States have shown the same results; and the Italian isotope study found that when the isotopic ratio of lead in gasoline was deliberately changed, the ratio in people's blood lead levels began to change as well; the ratio was still changing when the isotopes in gasoline were switched back, but already indicated at least 6 μg/dL came from gasoline lead.

The perplexing omission from the Commission's report of discussion concerning the relationship between blood lead and gasoline lead is magnified further by references in its report to the lead paint problem in deteriorating housing in the US. Because a lead paint problem in apartment dwellings does not apparently exist in Canada, the Commission comfortably implies that additional steps to phasedown lead in gasoline are not required. As noted above, the Commission should have known that EPA's census tract data indicated clearly that lead paint exposure did not confound analysis and interpretation of the NHANES II results.

Some have argued that because recent blood lead levels in Canada in 1984-1985 are lower than blood lead levels in the US in 1978 (the mid year of NHANES survey), that EPA's recent action further reducing gasoline lead levels was necessary, but that further action in Canada is not. This ignores the main point of the NHANES II lead analysis, which is that lead levels were falling in the US due to the decline in gasoline lead use. Gasoline levels in 1984-85, when EPA decided to tighten its lead in gasoline rules, had fallen substantially from 1978, and using the well established relationship between gasoline lead and blood lead, EPA predicted that mean children's blood lead levels would be down to about 10 µg/dL. This is very similar to the recent results in Ontario; and all of the benefits of further reductions in gasoline lead level that are discussed in "The Costs and Benefits of Reducing Lead in Gasoline" are for reductions below that new baseline. The recently released figures on the mean blood lead levels for Hispanic children in 1983 (from the Hispanic HANES) confirm that US blood lead levels were down at or below 10 µg/dL in the mid 1980's, which is very similar to the Canadian levels. This is precisely the drop predicted from the relationship between blood lead and gasoline lead levels that was derived, when mean blood lead levels in children were 17 µg/dL.

While the Commission's principal charge was health, it did find time to discuss the cost of further tightening of the gasoline lead standard. It cites a number of estimates and concerns, mostly from industry, indicating that reduction to the new US lead level would cost several cents per gallon. It omits mention of the fact that lead leads to faster corrosive wear in mufflers, carburetors, engine parts, spark plugs, and that the cost of this wear is also several cents per gallon. The ignored EPA analysis of the proposal to tighten gasoline lead standards demonstrated, after extensive analysis, that the maintenance savings from switching to low lead fuel would completely offset the higher manufacturing costs! This analysis survived extensive scrutiny in formal rulemaking procedures in the United States, and indicates that the net cost to consumers of reducing the amount of lead in leaded gasoline is approximately zero.

This places the health effects avoided in an entirely different light, because the cost of avoiding those health effects is zero! The public policy question then becomes whether those free health benefits will be accepted, or whether they will be turned down! This is an entirely different question to be posed to the Minister than the one posed by the Commission's report!

6. Summary

It is not objectionable to pose policy questions in a rational framework, and to present the advantages and costs of different alternatives dispassionately. Unfortunately, the Commission's Interim and Final Report, as provided to us, have not made adequate use of available information bearing on these alternatives. It is ironic that the Commission's failure to recognize the adverse health effects of lead at "low" concentrations and its failure to recommend more stringent measures to protect public health were based upon faulty assumptions about costs. In addition, another singular inadequacy is evident in the Commission's report: the willingness to ignore the results of highly relevant studies, published in the peer-reviewed literature, which demonstrate adverse health effects of lead in children and adults at blood lead concentrations below 30 µg/dL. In the end, it turns out, the added health benefits to be achieved by Canadian citizens through lowering the amount of lead in gasoline can be achieved for all Canadians at no monetary cost!

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